HANDBOOK
ON
POLIOMYELITIS

BY

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PREFACE

In 1840 the first monograph on poliomyelitis appeared, from the pen of Jacob Heine of Constatt. The disease had by then acquired enough importance to justify the integration of the existing knowledge in a book. In the course of the last hundred years or so, repeated efforts have been made to synthesize in book form accumulated experience; particularly since the time poliomyelitis ceased to be a children's disease and became the worst of the contemporary crippling conditions, unbounded by race, age or social status.

The present book represents one more attempt to take stock of the relevant views now held on most of the main problems related to poliomyelitis. In planning a book of this type it is not infrequent to follow one of two alternative methods Either a single author attempts to cover the whole field of experience (an enormous and frequently unrewarding task, for it is difficult for a single person to write with equal authority on subjects extending from virology to the recovery of muscle power) or else the book is formed by a collection of personal contributions Despite many brilliant chapters such books may lack the unity and logical sequence which seem required in attempting to describe in a limited number of pages the present views of experts in each of the main fields of enquiry

Poliomyelitis as a whole may be somewhat arbitrarily studied in three main sections: namely, the virus — in the laboratory and the human body — and its spread through the community; the respiratory disorders responsible for the lethality of the disease; and the paralysis of the locomotor muscles with its consequences

It seemed reasonable that, provided each of the contributors was well acquainted with the work and ideas of the others, a booklet of this nature could be written by three authors each one mainly, but not exclusively, responsible for each of the three sections of the work.

We hope that despite the diversity of the ground covered we have been able to preserve the necessary sequence of thought which will allow the reader to follow through the pages of the book as if reading the history of an event which occurred in days gone by. Perhaps the happy time is not far distant when such a contribution will have, if anything, only historical interest.

It is my pleasant duty before ending this introduction to thank my two collaborators for their interest in keeping their chapters as up to date as possible. I also thank Dr. M. Agerholm for the way in which she has carried out the task of editing the pages of this monograph.

J. TRUETA.

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We should like to express our gratitude to the many persons who have freely shared with us their knowledge and experience of particular aspects of the poliomyelitis problem.

Special contributors at the Nuffield Orthopaedic Centre have been Miss D Chapman, Miss A. Martin and Miss V. Smith, who as ward sisters have carried much of the increased burden of poliomyelitis in recent years; Mr. Trafford and Mr. Davis, who as hospital engineers have given unfailing aid in all problems of respiratory apparatus; Mr Salt, from the Nuffield Department of Anaesthetics, who has promptly solved for us a miscellany of problems of intubation and suction; Miss Isobel Ainger, whose twenty-five years' observant experience of rehabilitation in poliomyelitis provides a wealth of information invaluable to more recent en- [illegible]

respiratory complications, Miss Mary Kennedy, whose experience [illegible]

living for patients with upper limbs severely disabled by poliomyelitis; and finally the patients themselves who have helped considerably by analysing their own viewpoint and by posing for the illustrations.

From amongst our colleagues we should like to thank Dr. Kemp and Dr. Ardran of the Nuffield Institute for radiological help with the analysis and management of abnormalities of swallowing and respiration, Dr. Crampton Smith for his help as bronchoscopist and adviser on the problems of intermittent positive pressure respiration: and Mr Eric Peet of the Nuffield Department of Plastic Surgery for surgical closure of obstinate tracheostomata

Finally our thanks are due to Miss Arnott and Miss McClarty for the drawings; to Miss Bird for the photographs; and to Miss Pamela Dixon and Miss Ann Macbeth for secretarial help.

INTRODUCTION

Acute poliomyelitis is an infectious disease caused by any one of the three viruses of the same name. It is characterised by an acute inflammation of the central nervous system and its meninges. One type of nerve cell, the cell of the lower motor neurone, situated in the anterior horns of the spinal cord and brain stem, is particularly affected. It is the selective damage to these cells which causes the characteristic feature of the disease: a lower motor neuron paralysis, which, if the damage is irreversible, is associated with changes in the denervated muscles.

Infection with a poliomyelitis virus appears to be self-limited; it leaves behind a useful immunity against strains of the same type. "Chronic" poliomyelitis is a misnomer; it refers not to chronic infection or inflammation but to the lasting sequelae of the initial attack.

The term "poliomyelitis" is taken from the Greek (*πολιός* = grey; *μυελός* = marrow) and means inflammation of the grey marrow.

HISTORICAL REVIEW

Acute poliomyelitis is not a new disease. It is believed to have existed in ancient times. Descriptions of the disease — a flaccid paralysis of one or more limbs, occurring in children, without sensory impairment, often preceded by a short febrile attack, and followed by increasing deformity, — appear first in the medical literature of the late eighteenth and early nineteenth centuries, referring to cases developing in places as far apart as England, Italy and India ([1,2,3]). The pathology was described in 1856 by Duchenne, who correctly related the paralysis and wasting of the limbs to the "acute inflammatory atrophy of the ganglion cells in the anterior horns of the spinal cord," and observed that the initial attack could occur in adults as well as in children ([4]). Jacob von Heine wrote a monograph on the disease in 1860 ([5]).

What does appear to be new about the disease is its epidemic character. Severe epidemics, such as were experienced first in Scandinavia and the United States of America and have occurred more recently in populations all over the world, are so strikingly

characteristic and arouse so much fear that it is unlikely that they could have occurred in the past without being recorded.

The first report of an epidemic of poliomyelitis appears to be that by Bell, who wrote in 1831 — at second hand — of an epidemic which occurred in St. Helena: "an epidemic fever spread among all the children in the island about three or five years of age ... It

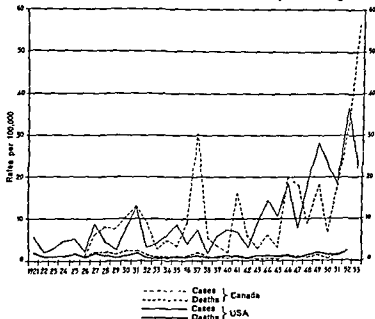


Fig 1 (a) Canada and U S.A
(notified deaths from poliomyelitis also shown)

Annual rates per 100,000 population of notified cases of poliomyelitis, 1921-1953 (Incidence of Poliomyelitis since 1920 M J Freyche and J. Nielsen, from "Poliomyelitis" W H O, Geneva 1955) *Note* the different scales on which these rates have to be presented

was afterwards discovered that all the children who had the fever were ... affected with a want of growth in some part of their body or limbs"(6). In 1843 an epidemic of paralysis in Louisiana, U.S.A., was reported — also at secondhand — by Colner· "The parents, (who were people of intelligence and unquestionable veracity) told me that eight or ten other cases of either hemiplegia or paraplegia (sic) had occurred during the preceding three or four months within a few miles of their residence, *all of which had either* completely recovered, or were decidedly improving. The little

sufferers were invariably under two years of age, and the cause seemed to be the same in all — namely, *teething*." (7). In 1876 C. F. Taylor, in the United States of America, in a monograph on "Infantile Paralysis and its Attendant Deformities" reported that "there seems to be no doubt that this disease is much more frequent now, and in this country, than formerly and is rapidly increasing" (8).

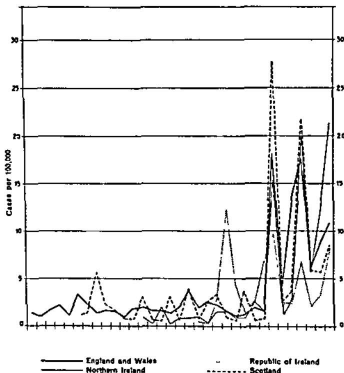


Fig 1 (b) British Isles

But there seems to have been [illegible] [illegible] [illegible] beginning of the twentieth centuries began to attack adults as well as children in places as far apart as Northern Europe, North America, and Australia. Such epidemics have spread steadily to

developed areas of Africa poliomyelitis is now "one of the most serious disease hazards faced by Europeans" [14].

Three striking features have emerged from the epidemiology of the disease in the twentieth century:

(1) the presence of *endemic* poliomyelitis in a population does not necessarily protect it against *epidemic* poliomyelitis.

(2) once the *epidemic* form of the disease appears in a community it must be regarded as forewarning of further (and often worse) epidemics to come.

(3) however the *endemic* form of the disease may be transmitted, the pattern of spread of the disease in *epidemics* resembles that of other infectious diseases transmitted by personal contact (fig. 2) [15, 16, 17, 18, 19].

Most of the epidemics which have been reported can be explained on one or both of two assumptions:

(a) the introduction into a population of strains of an unfamiliar type of the virus.

(b) an increased virulence of certain strains causing an increased incidence of frank disease per immunising infection.

When increased virulence occurs in strains of a type of poliomyelitis virus familiar to the population — in other words, endemic in it — the frank cases are confined to the younger age groups; their elders will have already acquired a predominantly symptomless immunity. When, however, the type is unfamiliar, all age groups are affected (if equally exposed) [20]. Both these patterns were illustrated by an epidemic affecting two contrasted populations in Malta in the winter of 1941—42. Of the 425 Maltese cases 98 % were aged 10 years and under (the majority under four years) and the Maltese adults, though equally exposed with their children, appeared to be immune. The immunity of the Maltese adults, however, contrasted strikingly with the susceptibility of the Servicemen from Britain — where the disease was also supposed to be endemic — of whom 55 developed poliomyelitis [21]. Such a distribution of frank disease would be explained by an enhanced virulence of the local virus which increased the hitherto low ratio of frank disease to symptomless immunising infection in those exposed for the first time to that type of virus — namely the new entrants to the population by birth and immigration, the Maltese children and the British adults (British children were not exposed)

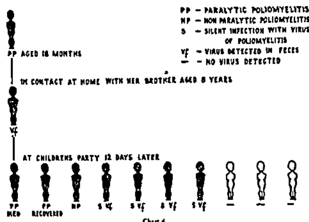


Chart 4

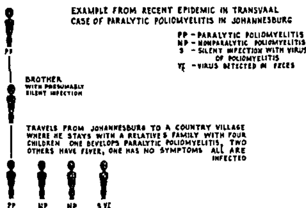


Chart 5

Fig 2. Diagrams showing the dispersal of poliomyelitis virus among contacts when

(a) the sister of a case attended a children's party Seven of the ten contacts studied were found to be carrying virus, three of them were symptomless It would be interesting to know whether the three contacts who did not yield virus were resistant to infection because of previously acquired immunity.

(b) the brother of a case travelled to a new family group which in its turn became a potential source of virus to the neighbourhood The importance of controlling the movements of contacts is obvious.

(J H S Gear and V Measroch "Poliomyelitis in Southern Africa". In Papers and Discussions of Second International Conference on Poliomyelitis Philadelphia, 1952)

study of the viruses and their antibodies was greatly accelerated. One of the first results of growth of the virus in tissue culture was the demonstration that the virus could multiply in and was pathogenic for cells other than nerve cells. Various tissues have been successfully used as culture media: human and monkey kidney and testis are particularly suitable. More recently "HeLa" cells from cultures of an epithelial carcinoma have been introduced for use in countries where monkey tissue is scarce (29).

Three types of poliomyelitis virus have been distinguished Type I (Brunhilde), Type II (Lansing), and Type III (Leon) All three can produce the same pathological disease, but each is distinguished by its distinct immunogenic character: infection with a strain of any one type stimulates in its host an immunity which protects against infection with further strains of the same type, but not against strains of either of the other two types. The demonstration of these three types in 1951 (30) provided an essential piece of information for the understanding of the epidemiology of the disease since it showed that, immunologically, poliomyelitis is not one, but three, diseases, and that to be immune to poliomyelitis *an individual or a population must acquire not one but three separate immunities.* A number of second, but not of third, attacks have been reported in the literature. Epidemiologically *it is obviously profitless to discuss immunity or susceptibility to "poliomyelitis" without specifying the type or types under discussion.* As a reminder of this important fact we have, where *relevant, used the plural "poliomyelitis viruses"* in place of the more usual singular.

Within each immunological type, as has already been suggested, *individual strains appear to vary in their virulence,* more virulent strains giving a higher incidence of frank disease per immunising infection, and the less virulent a very much lower incidence. *Presumably the less virulent strains are responsible for* the widespread subclinical immunisation to one or more types known to [illegible] (31, 32) and the more virulent strains for [illegible] h the [illegible] rank disease. The present world-wide increase in the incidence of *poliomyelitis (the disease as opposed to the virus)* would seem to be an expression, at least in part, of an increased proportion of strains of high virulence.

THE VIRUS IN THE BODY

The poliomyelitis viruses are found in the pharynx and pharyngeal secretions, the blood, and the gut and faeces of cases and of their contacts; they are also found in the central nervous system and lymphatic tissues of fatal cases. (33 34 35).

The period of pharyngeal carriage appears to cover 5 to 6 days before and up to 12 days after the onset of symptoms, and a comparable period in the symptomless case (36). It appears to correspond with the subject's greatest period of infectivity.

The period of faecal carriage is longer than the period of nasopharyngeal carriage in both frank and symptomless cases and may last as long as 12 weeks after infection, although probably as many as 80 % of infected individuals are free after 6 weeks (33). Chronic carriage of poliomyelitis viruses has not been shown to occur, and the presence of virus in pharynx or faeces may be assumed to indicate that the individual is passing through either an immunising infection, which may or may not develop into frank disease (see below, factors precipitating frank disease), or a "booster" infection, during which he is simply a temporary carrier.

Virus is found in the blood of cases in the preparalytic stage of the disease and in the blood of symptomless contacts at a similar time interval after exposure. By the time paralysis has developed the viraemia can no longer be demonstrated and the antibody titre of the blood has usually already begun to rise. Virus can, however, still be isolated from the central nervous system when death occurs after the development of paralysis in the acute stage

Outside the body the viruses are found, as might be expected, in sewage — though they have disappeared by the final stages of sewage purification — and in polluted rivers They have also been found in a variety of flies (37) In all these the virus, unlike many bacteria, seems to occur passively, without multiplication. Poliomyelitis viruses have not been isolated from domestic animals.

The *antibodies* with which immunity against each type appears to be associated are present in the blood and in the pharyngeal secretions of individuals who have been knowingly or unknowingly infected with virus of that type (38); in the blood of the newborn infants of immune mothers (in a titre which falls to zero over the first four months of life), and in the tissues of the central nervous system of individuals and monkeys who have had the paralytic form of the disease, the titre being highest in the parts most severely

attacked. They have also been detected in the cerebro-spinal fluid of animals in whom a high titre of blood antibody has been developed by active immunisation ([39]).

In experimental animals there appears to be a quantitative relation between the level of antibody in the blood and immunity. At low levels the animal is immune to oral or even intravenous challenge of virus but not to intracerebral challenge. Immunity to intracerebral challenge is obtained only with considerably higher blood levels; it is interesting that at these high levels the antibody spills over the blood-c.s.f. barrier and can be found in the cerebrospinal fluid. Animals with low levels of antibody in the blood *sufficient to render them immune to oral challenge* — *in the sense* of being protected against the disease — can become reinfected with the virus and temporarily excrete virus in their faeces without other evidence of infection ([39])

Studies of the geographical distribution of antibody are still all too few, but enough have been done to show considerable variations in different population groups ([31, 40, 41, 42]). In the United States of America antibody to Lansing virus appears to be the most widespread while the distribution of antibody of the other two types appears to be more limited and at present associated with more frank disease. This may well be true of Great Britain also, since here, although sporadic cases have yielded Lansing virus, only strains of Brunhilde and Leon types have been reported as causing local epidemics. Until further studies are available the [illegible] of the British population remains a matter of [illegible]

susceptible to one or more of the poliomyelitis viruses.

TRANSMISSION

Since virus is present in both the nasopharyngeal secretions and the faeces of cases and their contacts it can theoretically be spread through the population by oral or bowel *transmission, i.e.* = like either diptheria or typhoid fever. In practice, in countries such as Great Britain where water supplies are usually protected from sewage contamination, the pattern of spread resembles more the spread of diphtheria or measles than of typhoid fever, i e. it appears to be by "personal contact" rather than by food and water contamination. In outbreaks where the source of infection of

cases has been carefully traced, personal contact with a case or an intermediate has been found in 70 % of cases, while attempts to incriminate food, water and flies have repeatedly failed (21). Infectivity appears to be limited to the first three weeks following infection. Contamination and infection by faeces, sewage and olluted rivers can, of course, also occur when standards of ygiene allow it, and an individual may acquire the virus from any or these less common sources.

Dosage of virus appears to play a significant part in determining whether infection occurs. Repeated or close contact with a case appears to be more dangerous than a brief social encounter. Schools, particularly nursery schools, seem to be especially favourable to transmission of the virus, probably for this reason, and a familiar epidemic pattern is that of a school as the radiating focus of infection, with adults and pre-school children affected only as they are close contacts of the school children.

Generally speaking, adults appear to be less exposed to infection outside their home than children. Non-immune adults, when exposed, are however, just as susceptible to infection as children —and, as already mentioned, they often get the disease particularly severely. This phenomenon occurs also in a number of other virus infections and is not peculiar to poliomyelitis.

The proportion of symptomless to frank cases appears to vary from place to place and in the same place from time to time. Ratios as high as 1 frank to 5 symptomless infections (13) and as low as less than 1 in 1,000 (21) and all intermediate grades (41) have been reliably reported

The ratio of subclinical infection to clinical disease in any one opulation group at any one time must be regarded as the product several factors, among which the following may be mentioned·

(*i*) the virulence for the nervous system of the infecting strains

(*ii*) the prevalence of conditions favouring transmission of the virus, e.g climate (humidity and temperature) and the activities of the population (outdoor, or indoor, gregarious, isolated, etc.) The main effect is probably on the *dose* of virus transmitted to the individual.

(iii) the immunological state of the population or of sub-groups of the population exposed to the virus.

(iv) the prevalence of conditions favouring the development of frank disease. (see precipitating factors, p. 17). Populations

subjected to courses of routine injections of irritating substances have on at least two occasions been shown to be strikingly susceptible to the disease (see p. 17). Similarly particularly susceptible sub-groups of a population are found in maternity homes (pregnant and lactating women and newborn infants), and in hospitals (the tonsillectomised and those receiving irritating injections).

OURSE OF THE INFECTION IN THE INDIVIDUAL: FACTORS PRECIPITATING FRANK DISEASE

e progress of the virus in the body is not yet fully understood, but the following probably represents most of the essentials.

Whatever the source of the virus it enters the body via the pharynx. If conditions are favourable, it remains and multiplies there for about 2 to 3 weeks and at the same time is swallowed and reaches the intestines, to be excreted also in the faeces for a longer period (from 4 to 12 weeks) at the end of which it disappears. During the first three to four weeks, viraemia occurs intermittently, and the clinical manifestation of the infection, acute poliomyelitis, may occur. Re-excretion of systemic virus into the pharynx and gastro-intestinal canal also occurs — as has been shown experimentally [44] and by transmission of the virus to the contacts of cases receiving live virus in faulty vaccine, given intramuscularly [45]. The appearance of antibody, which is usually detectable by the time clinical disease becomes apparent, presumably accounts for the disappearance of the virus, first from the blood and pharynx, and later from the stools.

In many cases this is the whole story: infection, viraemia, antibody production and elimination of virus from the body. The infection is silent and at the end of it the patient has acquired immunity against further infection with other strains of the same type, either without any symptoms at all or with symptoms so mild and indefinite that they would not be suspected as being manifestations of acute poliomyelitis unless the patient were a known contact of a frank case, or the possibility of such an infection was uppermost in the mind of the patient or his doctor, because of a local outbreak.

To cause the disease "acute poliomyelitis", non-paralytic or paralytic, the virus must invade the central nervous system. At one time it was thought to do so by travelling along nerve fibres,* but it seems unlikely that this route really gives the virus direct

* There is considerable evidence that the virus can travel up nerve fibres, in experimental conditions, but very little evidence that this route plays an important part in natural conditions.

access to the central nervous system, and the demonstration of virus in the blood in the pre-paralytic stage in humans and experimental monkeys, and in symptomless human contacts,[46] and the widespread distribution of the virus in the c.n.s. in frank cases suggests that the infection of the central nervous system is in many cases "blood-borne".

To reach the cells of the central nervous system from the blood the virus must cross the "blood-brain" barrier. This is a physico-chemical barrier which protects the nervous system from noxious substances circulating in the blood; its presence explains why the

some factors in precipitating frank disease appears to depend on this lowering of the blood-brain barrier; similarly the "virulence" of certain strains may be due to some property which enables them to cross the barrier.

Once in the central nervous system the virus enters the nerve cells and multiplies inside them, causing damage which is reflected clinically as loss of function. The virus does not appear to live in the cells for a long time before giving rise to symptoms and *probably not more than 12—48 hours intervenes* between the penetration of the cell and the appearance of pathology and paralysis[17]. The large motor neuron cell appears to be the most susceptible to the virus, since it alone may die, whereas virtually all the other *affected cells appear to recover. Were it not for the unexplained* susceptibility of this one type of cell the poliomyelitis viruses would have little more than mild nuisance value to their human hosts.

The incubation period of the frank disease varies *from a few days to* up to 3 weeks after exposure. In a study of contacts of cases in New York 96 % of secondary cases occurred within two weeks of the first case. In children the period was on the whole shorter than in adults, and the onset was practically confined to the first week following diagnosis of the primary case[18].

As the patient develops antibody the virus disappears from the tissues; it cannot usually be isolated from the central nervous system after the 12th day, but it is, of course, possible that it remains for a longer period inside infected cells where it cannot be reached by antibody. As already mentioned it can persist in the gut for anything up to 3 months from onset.

FACTORS PRECIPITATING FRANK DISEASE

There are at least four factors which have been found to increase the individual's chances of developing frank disease during his period of immunising infection:

i) *Trauma* to an area precipitates paralysis in the parts sharing the same segmental innervation as the traumatised area. The trauma may be any incident which causes a fairly prolonged inflammatory response, but particularly guilty have been intramuscular injections of irritating substances. An association between acute poliomyelitis and diphtheria immunisation has been reported from Australia, the U S.A. and Britain, and between acute poliomyelitis and intramuscular arsenical preparations on two occasions from populations being treated for yaws ([49, 50, 51]).

"Double event" cases, viz, an irritating injection or other trauma followed between one and five weeks by an attack of acute poliomyelitis (from which poliomyelitis virus can be isolated) differ from simple cases in that the paralysis begins, and is maximal, in the muscle group sharing the same segmental innervation as the traumatised area, sometimes the entire motor innervation of the injected limb appears to have been destroyed The paralysis is, however, not necessarily confined to this group and severe generalised paralysis and death may follow.

The mechanism of this phenomenon is not yet fully understood. The peripheral inflammation may cause a reflex vascular response in the relevant segments of the cord ([52]), so that there is a local increased permeability to a coincidental viraemia, or it may cause a reflex change [illegible]

chance of coinciding with what might otherwise have been a symptomless infection with poliomyelitis virus, and the reason why diphtheria immunisation has been particularly guilty in this country is presumably contained in the fact that it is performed at an age when many children pass through their first — usually symptomless — infections with strains of one or more of the three types of virus

The incidence of "double event" cases in any one community at any one time depends presumably on three main factors: prevalence of the virus, the incidence of the precipitating factor, and the immunological state of those exposed to both the first two

factors. Thus, the problem did not manifest itself clearly in Britain until 1947, a year when an effective diphtheria immunisation campaign coincided for the first time with a national epidemic of poliomyelitis and the London boroughs reported as many as 25 characteristic "double event" cases during the *first* six months of that year. [illegible]

quota of these characteristically severely paralysed children. The solution to the problem presumably lies in the correct sequence of routine immunisation, e.g. poliomyelitis immunisation before diphtheria immunisation, for it is to be hoped that the early development of poliomyelitis antibody following poliomyelitis vaccination will cancel out the precipitating effect of its local inflammatory reaction.

(ii) *Tonsillectomy* precipitates particularly the bulbar type of the disease which may develop as early as the second day following operation ([53]). This may have the same explanation as (i) or be due to the direct drainage of infected blood and lymph from the nasopharynx into the brain stem and upper spinal cord, or to a combination of both. [illegible]

recently ([54]).

(iii) *Excessive exercise and chilling* during the immediate "pre-paralytic", or viraemic, period appears to increase the chances of developing frank disease, and a relation has sometimes been found between the most exercised and the most paralysed limb. ([55] [56]) [illegible] imming is probably [illegible] ch swimming baths [illegible] These factors may have the same physiological basis as (i).

(iv) *Pregnancy and the period of lactation* appear also to be predisposing factors. In Denmark, in 1936, 20 of the 62 cases aged between 20 and 34 years were pregnant — a figure three times the expected incidence of pregnancy *in that age group* ([57]). Among 22 cases occurring in relation to a small school in southern England in 1952 the only fatal adult cases were a pregnant and a lactating woman respectively; (it is also interesting that the only fatal child case was a "post-injection" child) ([58]).

Individuals falling into any of the above four groups must be regarded as urgent candidates for passive immunisation with gamma globulin, when they are direct or indirect contacts of frank cases.

Pregnant women are also obvious priority candidates for active immunisation.

CHAPTER 4

THE PATHOLOGY OF POLIOMYELITIS

PRIMARY PATHOLOGY OF POLIOMYELITIS

The primary pathology consists in:

(i) *A generalised disorder of the central nervous system and meninges*, consisting of changes in a wide variety of nerve cells, and an associated inflammatory reaction (vaso-dilatation, haemorrhages and white cell infiltration). These changes are usually maximal in the spinal cord and brain-stem and occur mainly in relation to the distribution of the damage to the cells of the lower motor neurones [illegible]

be predominantly reversible, (except in very young infants, in whom permanent cortical damage has been reported (59).

Some, but not all, of the more general inflammatory changes seen at post-mortem in fatal cases are explained by the anoxemia in which the majority of these cases die, but animal studies have confirmed that the general changes throughout the central nervous system in non-fatal and even in non-paralytic cases are usually more severe and more widespread than clinical evidence would suggest.

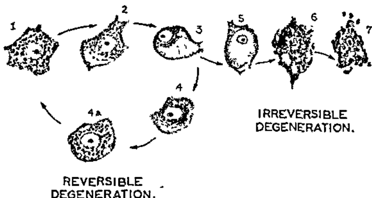
[illegible] most [illegible] ch [illegible] or horns of the grey matter where they may be concentrated at one level or on one side of the spinal cord, or be patchily distributed.

(ii) *Damage to the cells of the lower motor neurons, and disruption of the motor unit.*

The large motor cells of the lower motor neurons show every grade of change from mild reversible chromatolysis to irreversible degeneration and death, after which they either disappear or remain as shadow cells embedded in glial tissue (fig. 3). Destruction of large numbers of motor cells causes a microscopic shrinkage of the cord at the levels affected.

When the motor cell dies the changes are not confined to the spinal cord, but involve the whole motor unit of which the cell body is the central part. It is the pathology of the denervated muscle

fibres of the affected motor units which is responsible for much of the deformity occurring as a sequel to paralytic poliomyelitis, particularly in children. This peripheral component of the pathology of poliomyelitis is so often forgotten that a brief account of the motor unit and the changes occurring in it following death of the cell may not be out of place here. Understanding of the anatomy and function of the motor unit is essential to the management of poliomyelitis in all its stages.



1 Normal cell, central nucleus, strongly staining Nissl bodies
2 Nissl bodies paler than normal
3 [illegible]
4 [illegible]
4a [illegible]
5 [illegible]
6 Nucleus pnykotic cytoplasm granular with vacuoles.
7 Beginning neuronophagia

Fig 3 Diagrammatic presentation of the changes which may take place in a motor nerve cell infected with poliomyelitis virus

THE MOTOR UNIT (fig. 4) A motor unit consists of

(a) *a motor cell* situated either in the anterior horn of the grey matter of the spinal cord or in one of the motor nuclei in the brain stem;

(b) *a myelinated axon* which runs out from the cell body via an anterior root, peripheral nerves and intervening plexuses, without any synaptic interruption, to enter the substance of a muscle, where it breaks up into the number of end branches, each of which ends on one muscle fibre;

(c) *the muscle fibres* related to these end fibres of the axon.

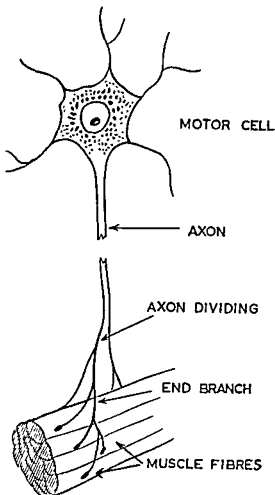


Fig 4. Diagram of a motor unit.

The muscle fibres of a single motor unit lie in close anatomical relation to one another. Each appears to receive only one end branch, and to belong therefore to only one motor unit. The number of muscle fibres in any one motor unit appears to be related to the delicacy of adjustment required of the muscle; thus in the eye muscle the number is small, (the ratio of muscle fibre to axons entering the substance of an eye muscle may be as low as 2 : 1 or even 1 : 1), while in muscles in which only coarser adjustments are

required, such as the glutei and the quadriceps, the ratio is very much higher, and one axon may innervate as many as 140 muscle fibres. *All* the muscle fibres of one motor unit respond simultaneously to an impulse transmitted by the motor cell or stimulated artificially anywhere along the course of the axon: in other words the motor unit shows an "all-or-none" response; if the stimulus is sufficient to induce a contraction, all the muscle fibres of the unit contract simultaneously; if the stimulus is insufficient, none contract. The different degrees of force obtained by voluntary or reflex contraction of a whole muscle appear to be obtained by variation of the number of such motor units brought into simultaneous action, in other words, by variation in the number of cells stimulated in the cord.

The muscle fibres depend intimately on their contact with the motor cell, via its axon, not only for voluntary or reflex contractility but also for their normal anatomical and physiological state. The motor unit is therefore a physiological unit and not simply an anatomical relation. Disruption of the unit, either when the cell dies and its axon degenerates (as in poliomyelitis) or when the axon alone degenerates (as in injuries of the nerve roots and peripheral nerve) is followed by both anatomical and physiological changes in the muscle fibres thus "denervated." They shrink, [illegible]

[illegible]

the elasticity of the muscle as a whole. This has the effect of "freezing" the muscle at the length at which it is held — or at the maximum length to which it is repeatedly stretched — during the development of the "denervation changes". These changes are the basis of the development of contractures and hypermobility during the period of denervation following poliomyelitis or other conditions causing degeneration of the axon.

The duration of the period over which true denervation changes occur is not clearly defined Probably it is complete by the end of the first four months. After that time the tendency to develop either contracture or excess length of the muscle has usually ceased

in the adult. In the child the tendency persists throughout growth, as the pathological muscle causes an imbalanced pull on the muscles and growing bones, and it can be responsible for late deformities of trunk and limbs developing even many years after the initial attack. It is unfortunate that the period during which denervation changes occur most rapidly, corresponds with the period in poliomyelitis in which it is often most difficult to ensure optimum length of the muscle concerned, for both (i) "spasm" (see below) and (ii) the postural demands of the respiratory and bulbar paralysis may make the task of avoiding deformity difficult, and while at that time one may not be able to forecast the ultimate paralysis and so choose which "contractures" to encourage — for not all contractures are bad.

Pathology of "spasm"

The pathology of so-called "spasm" in poliomyelitis should be considered here, since its correct management depends on an understanding of its pathology.

The word "spasm" is used in poliomyelitis to describe the involuntary contraction of muscle which occurs in both the acute and the transitional (see below) stages of the disease. It may occur even at apparent rest, or be precipitated by passive movement of the part. It should not be confused with the isolated brief twitching of muscles occasionally seen in the earliest days of the infection before paralysis can be detected, nor with the "spasticity" of upper motor neuron lesions.

"Spasm" seen in the first few weeks of the disease is predominantly meningitic in origin. The inflammation of the central nervous system and the meninges makes any movement which stretches them acutely painful, and in consequence there is a reflex contraction of the muscles preventing their stretch. This is seen in the [illegible]

hips and knees and plantar flexion of the foot). If the muscles concerned are totally paralysed the "spasm", and that particular part of the characteristic posture for which it is responsible, is, of course, absent. The movement which it would prevent is, however, still painful to the patient and it will still stimulate any surviving motor cells to attempt to transmit impulses, although no effective

resistance may be felt. The importance of the pain and the "spasm" it produces is obvious: if exercise is harmful in the presence of virus measures to reduce pain and spasm may not only reduce the discomfort and deformity but also the amount of motor cell damage.

As denervation proceeds a peripheral cause of "spasm" supervenes — the result of the denervation shortening already described. Stretching of the already shortened muscle pulls on and tears newly-formed fibrous tissue, and any residual innervated muscle fibres respond by contracting to protect it.

Another cause of resistance to stretch — which is not spasm, since it is not contractile — is also encountered. This is the rigid resistance of the fully formed fibrous tissue in denervated or partially denervated muscle; it is felt as a hard, unyielding band. The rigid anterior and posterior axillary walls found in a patient in whom denervation atrophy has occurred with the arm in the adducted position are common examples.

CHANGES IN THE SYMPATHETIC NERVOUS SYSTEM

There is a considerable amount of evidence of involvement of the cells of the sympathetic nervous system in acute poliomyelitis (60). Both the cells of the sympathetic nuclei in the dorsi-lumbar segments and the post-ganglionic cells of the sympathetic chain have been reported to show histological changes, and correspondingly both hyperhidrosis and anhidrosis have been reported clinically. Hyperhidrosis suggests a release phenomenon of the cells in the sympathetic chain due to death of the related cells in the cord; anhidrosis or more commonly hypohidrosis suggest death of post-ganglionic cells in the chain. The circulatory conditions most characteristic of poliomyelitis, namely cold, blue extremities, chilblains and impaired growth also suggest sympathetic involvement (overactivity) but in practise it is difficult to distinguish how much they are due simply to the inactivity and denervation changes in muscles and how far to true sympathetic dysfunction.

SECONDARY PATHOLOGY

CHANGES IN THE LUNGS. These are the result of the underventilation and consequent stasis of the lungs, or of the aspiration of saliva and vomit and retention of secretions which occur when there is an associated respiratory or "bulbar" paralysis. The characteristic findings are (i) *atelectases*, or collapse of parts of the lung, (ii) *oedema*

with a fluid level determined by the posture of the patient at the time, (iii) secondary infection. They are discussed more fully in Chapter 8.

CHANGES IN THE URINARY TRACT. These can be caused by retention of urine resulting from the initial transient paralysis of micturition

of temporary urinary retention or prolonged recumbency.

CHANGES IN THE SKELETON. Skeletal abnormalities occur almost exclusively in those who develop their paralysis while still growing. They fall into two groups: on the one hand, reduced growth producing shortening of an affected limb sometimes with early epiphyseal closure; on the other, distorted growth resulting from imbalanced muscle pull on the growing bones.

CHAPTER 5

CLINICAL DESCRIPTION, DIAGNOSIS, AND DISPOSAL

Acute poliomyelitis is a self-limiting virus infection, which may be symptomless or may cause symptoms ranging from a mild feeling of "being off colour" to a generalised encephalomyelitis with meningism and lower motor neuron paralyses and their complications. The clinical picture is described here:

Onset

The onset of the disease may be either rapid or gradual. Cases with rapid onset may wake with fully developed paralysis after going to bed with only the mildest symptoms the night before, and it has been known for a child to go to bed with only mild pyrexia and be found dead from bulbar paralysis the following morning. Most often, however, the onset is gradual, and, particularly in adults, may be divided into two distinct phases, the minor and the major illnesses, which may merge or be divided by an interval of anything from two to five days, during which the patient feels almost or quite well and may return to his normal activities.

The minor illness

The usual symptoms of the *minor illness* stage of the disease may be either *influenzal* or *gastro-intestinal*. In the *influenzal* type the main symptoms are headache, aching of the limbs, tenderness of the muscles, shivering and fever (which may be up to 102—3° F.). Mild sore throat may be complained of, but coryza and coughing are rare. In the *gastro-intestinal type* sudden vomiting, often unaccompanied by other gastro-intestinal symptoms, is a common manifestation, particularly in young children, but almost any combination of symptoms suggestive of "gastric flu", "summer diarrhoea", or dietary indiscretion may occur.

At this stage the symptoms and signs may (a) remit altogether as the patient recovers from the minor illness without the involvement of the central nervous system which constitutes the major illness; or (b) remit for only a few days before the same symptoms recur in a more severe form; or (c) they may pass straight on into the major illness without remission.

THE MAJOR ILLNESS

Headache is usually present, is usually severe and is often described as "the worst headache I have ever had.". There may be *vomiting*, sometimes projectile and repeated, diarrhoea or constipation. There may also be *hyperaesthesia* or *paraesthesiae* (often in the fingers), but usually the generalised aching and malaise of the minor illness becomes particularly concentrated in the head, neck and back, and resistance to passive flexion of the neck and spine develops.

There is nearly always some degree of *meningism* unless the central nervous involvement is very localised. Neck stiffness can be severe, but rarely proceeds to the head retraction seen in other conditions. Useful tests for the relatively mild meningism of poliomyelitis are·

(a) the *"kissing the knees"* test (see fig. 5). If a child does not co-operate, passive imitation of the movement with the child lying on his side may reveal pain on flexion of the spine and neck.



Fig 5. "Kissing the knees" test. *Bilateral* failure to approximate knee and mouth and resentment of attempts to perform the movement passively is a useful early sign of meningeal irritation

(b) the *"straight leg raising"* test (see fig. 6). This is usually positive bilaterally except in cases in which only the upper part of the cord is involved. Dorsiflexion of the ankle performed when the straight leg is held just below the angle producing pain, is used to confirm that the pain is due to traction on the sciatic nerve and its roots and not to pathology outside the central nervous system.

(c) the *"head-dropping"* test. When the child is lifted off the bed by the armpits the normal active simultaneous flexion of the head is absent and the head falls back.

(d) the *"tripod"* sign (see fig. 7). When the patient *is sat up* (this should not be done in cases suspected of bulbar involvement) he puts his arms behind him for support because he cannot tolerate the flexion of his spine, which occurs in the sitting position.

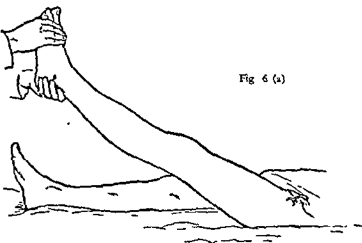




Fig 6 Straight leg raising test. The straight leg is lifted off the bed by the heel
(a) with the foot in plantar flexion
(b) in the neutral position or dorsiflexion.
In the second position the leg cannot be lifted so far

At this stage, before frank paralysis occurs, careful neurological examination may reveal evidence of encephalitis and of early lower motor neurone involvement.

The encephalitis may have mainly cortical or mainly cerebellar manifestations. The tendon jerks may be increased and the plantar

response extensor, but these signs are usually transient and are soon replaced by diminution of reflexes and absent plantar response — usually asymmetrical at first — as the lower motor neurone involvement predominates. A fine nystagmus is a common early finding, and there may be ataxia and tremor not explainable by the motor weakness.

Early lower motor neurone involvement is not always easy to detect. Obvious weakness is usually late both as a symptom and as a sign. Thus a patient rarely complains of weakness as such in the early stage; he is more likely to say that a limb feels heavy or numb or stiff and his first recognition of weakness is often when his legs "give way" on getting out of bed — a sign too often attributed by observers to dizziness or even a faint. Similarly, actual weakness of a muscle or group of muscles can probably not be detected even by

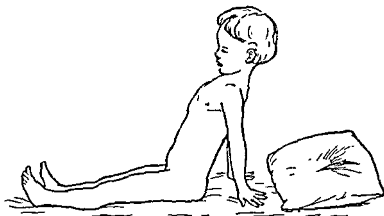


Fig. 7. Tripod sitting. The stiffness of the back prevents the normal flexion of the spine on sitting up, and the child has to support himself with his hands behind him. (*Not* to be tested if failure of swallowing is feared)

most careful clinical examination, before about 50% of the neurons concerned are out of action[62]. The earliest sign of lower motor neuron involvement in poliomyelitis is diminution or loss of both superficial and tendon reflexes; it may appear many hours, or even several days, before actual weakness is detectable, or it may never proceed to obvious weakness. The importance of testing the reflexes of any child with unexplained fever who is too young to perform voluntary movements on request, should be emphasised.

Other early indications of paralysis are:

(i) The intermittent or progressively nasal speech of early palatal palsy.

(ii) [illegible]

(iii) [illegible] patient who wishes to clear mucus from his throat but dare not inspire sufficiently to give effective clearance.

(iv) Reluctance to talk or shortness of breath on talking of a patient with early respiratory paralysis

(v) Unequal expansion of different parts of the chest without underlying physical signs of pulmonary disease.

(vi) Absence of the normal contraction of the abdominal muscles as the patient tries to lift his head or sit up in bed.

(vii) Abnormal posture of a limb due to loss of tone, e.g. external rotation of the whole lower limb, or increased plantar flexion of the relaxed foot.

(viii) Tricks to move a limb, e g. helping one limb with another, "carrying" one lower limb on the foot of the, as yet, spared limb; flexion of the hip with the hands.

Characteristic features of the *frank paralysis* when it does develop are that it is at first patchy, is often asymmetrical, and tends to affect more severely the proximal rather than the distal muscles of the limbs Even when paralysis is very extensive it is not uncommon for some power to be preserved in the fingers or toes. When weakness is present the reflexes are always more reduced than the power.

At this stage of the disease, however, no good purpose is served either by searching too minutely for weakness or by exhaustive muscle charting. If a muscle is not obviously and definitely weak, it is not wise to hang too much in the way of diagnosis upon its weakness, which may well be due to inhibition by pain from other causes. In any case considerable fluctuations in paralysis usually occur and the muscle findings will very probably be different the next day There is therefore no point in needlessly fatiguing a patient when, if the condition is poliomyelitis, fatigue is one of the known factors in aggravating paralysis.

If poliomyelitis is suspected, however, there are some specific points to which attention must be paid These are:

(i) palatal palsy, because it suggests an incipient swallowing defect;

(ii) the swallowing defect itself;
(iii) laryngeal stridor;
(iv) weakness of the deltoid because of its value in forecasting respiratory complications; the segmental innervation of the deltoid (C. 5 and 6) is at a strategic position from which the virus and paralysis spread either up to the phrenic outflow and bulb, or down to the thoracic segments;
(v) diminished or asymmetrical respiratory excursion and the use of accessory muscles of respiration.

Examination other than neurological usually reveals no signs characteristic of poliomyelitis. A rising pulse rate and blood pressure (associated with carbon dioxide retention and hypoxia) indicate significant impairment of respiratory function.

LABORATORY FINDINGS IN POLIOMYELITIS.

Routine examination of *blood* and *urine* show no features typical of poliomyelitis. The blood cell count and blood sedimentation rates are normal.

The *cerebro-spinal fluid* shows only non-specific changes on lumbar puncture. *The pressure may be slightly, but not greatly, raised.* The fluid appears clear and colourless; its cells and biochemistry may be quite normal, particularly if lumbar puncture is done early in the disease, but usually they show some changes characteristic of a mild meningism: an initial moderate polymorphonuclear leucocytosis is soon succeeded by a lymphocytosis. The number of cells varies from 20 to 200 cells/cmm. The cell count returns only slowly to normal over several weeks.

The protein is usually slightly raised in the acute stage, gradually climbs to about 200—300 mgm/100 cc., rarely more, and then gradually falls during the following months. Sugar and chlorides *remain within normal limits, unless there has been excessive* vomiting, in which case the chlorides may be lowered.

The non-specificity of the changes in the cerebro-spinal fluid [illegible]

[illegible] spinal fluid does show a characteristic picture, and in which there is a specific therapy which must be initiated early. Lumbar puncture [illegible] increase paralysis; until [illegible] t be regarded as [illegible] hen there is real

doubt that the condition may not be poliomyelitis, but some other condition in which early diagnosis is important for its treatment.

Isolation of the virus from blood, nasopharyngeal and faecal specimens is not yet a routine clinical procedure. Similarly, estimation of antibody to show a rising blood titre could be a useful diagnostic aid, but would not help in the early stages. A complement fixation test has been introduced recently but is also not yet a routine procedure.

DIFFERENTIAL DIAGNOSIS

The clinical picture of poliomyelitis differs so markedly at its [illegible]

from other febrile conditions; *the non-paralytic stage of the major illness* must be distinguished from other conditons causing meningism, and the *paralytic stage* must be distinguished from other conditions causing lower motor neuron paralysis.

(a) Differential diagnosis of the minor illness stage

From the description given it will be seen that the possibility of poliomyelitis has to be considered at the onset of a number of widely differing febrile conditions. The likelihood of poliomyelitis is suggested if there has been a unusual amount of "minor" illness occurring in the family or institution at the time; a frank case of poliomyelitis in a school is often preceded by what the staff will tell one is a quite unusual incidence of minor illness, not characteristic of any particular disease. Similarly, a frequent story is of trivial illness (unexplained headache, sudden vomit, brief fever) in the family or its contacts before the actual clinical case finally developed perhaps in an individual who was particularly susceptible, e.g. a post-inoculation child or a pregnant or lactating woman. It must, however, be remembered that isolated cases do occur, particularly in children, and that, though the disease is commonest in the summer and autumn, there is no time of year and no age group in which acute poliomyelitis does not occur.

Streptococcal and diphtheritic throat. The local signs are more [illegible]
ment [illegible]
may [illegible]

elicit that the complaint is rather of *difficulty* than of *pain* on swallowing. Testing for swallowing defect (see p. 70) will indicate the significance of the complaint.

Influenza. The temptation to diagnose "flu" out of the winter months, when the influenza virus is virtually absent from Great Britain, should in itself suggest the possibility of poliomyelitis. "Summer 'flu" is one of the commonest early misdiagnoses by both patient and doctor. Coryza, tracheitis and bronchitis are not common in poliomyelitis.

Gastro-intestinal disorders. A striking differentiating feature is often the disproportion in symptoms in poliomyelitis e.g. vomiting without abdominal pain or diarrhoea, too high a fever for the relatively mild abdominal signs, etc. The classical triad of anorexia, abdominal pain and constipation has led to more than one patient with acute poliomyelitis having his appendix removed; careful examination with both conditions in mind should prevent this error. Rectal examination will help to differentiate the tenderness of the abdominal muscles from tenderness of visceral origin.

Typhoid fever may present with a picture similar to early acute poliomyelitis, but in typhoid the fever is usually higher, the patient more ill, the tongue drier than in non-paralytic poliomyelitis. Blood and stool examinations will differentiate the conditions.

(b) Differential diagnosis of the meningitic (pre-paralytic) stage of the major illness

Here diagnosis depends on exclusion:

The suppurative meningitides: The patient is usually more ill, less alert, more confused than the patient with acute poliomyelitis who, unless already anoxic, is characteristically bright compared with his signs of illness The characteristic petechiae will often suggest the diagnosis of meningococcal meningitis. The cerebro-spinal fluid, which may be cloudy, shows a greater polymorphonuclear leucocytosis, lowered sugar and raised protein, and the organisms will be seen on smear or culture. Blood culture may be positive.

Tuberculous meningitis: The textbook case of tuberculous meningitis has a history of several weeks' indefinite illness culminating in meningeal symptoms; a history of contact with an open case may be elicited; radiography shows involvement of the lungs; tubercles are seen in the eye fundi; lumbar puncture yields a lymphocytic fluid with marked reduction in both chlorides and sugar and the bacillus

is seen with Zeehl-Neilsen staining. But, although all these signs should be looked for in a suspected case, some or all of them may

poliomyelitis, the clinican must use all his skill and resource to decide which of the two conditions is really the more likely,

Othe glandular or a Cocks With increasing facilities for isolation of virus by tissue culture it has become obvious that a number of cases of so-called "non-paralytic poliomyelitis" are in fact due to either a Cocksackie or one of the so-called "orphan" or unnamed viruses.

The encephalitides: A history of recent vaccination or a recent

Other causes of neck stiffness. Local inflammation round the neck, and early apical pneumonia, which may present with a localised stiff neck, may have to be excluded. The limitation of the stiffness to the neck, absence of associated back stiffness, and a negative "straight leg raising sign" suggest other causes than true meningism.

(c) DIFFERENTIAL DIAGNOSIS OF THE PARALYTIC STAGE

Pseudoparalysis caused by inflammation of the related bony or soft tissues which inhibits movement of the limb may well simulate poliomyelitis *Acute osteomyelitis, septic arthritis, acute rheumatism, inflamed glands* and *scurvy* have all presented diagnostic problems. Careful local examination, however, will usually reveal the local cause of the patient's reluctance to use the limb or limbs, and laboratory examination the aetiology. The sedimentation rate is a useful guide; it is normal in acute poliomyelitis and raised in the acute septic conditions and acute rheumatism.

Hysterical paralysis is not uncommon, particularly in poliomyelitis

outbreaks. Routine neurological examination and the preservation of the tendon reflexes in a so-called paralysed limb, will usually help to convince both the patient and his doctor of the absence of true weakness. However, one must not forget that clinical examination in an unco-operative patient is a very poor measure of muscle power; occasionally the patient who fails to justify his symptoms by gross weakness is told to "snap out of it" only to present a recurrence of symptoms and undoubted paralysis after resuming his normal activities. It is therefore probably wiser, particularly if the patient is slightly "off colour", has had mild fever, or is a contact, i.e. where there is reasonable doubt, to give him the benefit of it and let him idle for a couple of weeks until he is out of the danger period, making sure that he is suitably reassured in the meantime.

Upper motor neurone paralyses. Increased reflexes, a positive "Babinski" sign, and increased tone indicate that the weakness is of upper and not of lower motor neuron origin. The presence of protective "spasm" in poliomyelitis (see p. 24) may occasionally suggest the spasticity of an upper motor neuron lesion, but careful analysis of the muscles involved, the demonstration that only certain movements elicit spasm, the contrasting loss of tone in neighbouring muscles, the asymmetry of the reflexes, the absence of an extensor plantar response, and the patient's complaint that the resisted movements cause pain along what is obviously the course of the nerve trunk, all help to distinguish spasm of meningitic origin from the spasm and increased tone of an upper motor neuron lesion.

Paralyses in the "acute polyneuritis" group. This term covers a number of quite different pathological processes, ranging from the Guillain-Barré syndrome, (with sensory abnormalities, paralysis maximal distally and the characteristic cell-dissociation in the cerebro-spinal fluid) via the paralyses caused by rare viruses, specific toxins (e.g. agricultural pesticides, shellfish poisoning) biochemical disorders (e.g. potassium deficiency paralysis) and perhaps individual allergic conditions. If the patient is first seen with respiratory embarrassment or in coma diagnosis may be a real problem. The following are useful distinguishing features, one or more of which may suggest that the diagnosis is *not* acute poliomyelitis:

(a) the history may show a gradual onset, possibly with preliminary sensory symptoms, e.g. glove and stocking paraesthesiae or anaesthesia;

(b) there may not be the characteristic fever;

(c) there may be demonstrable sensory impairment — again, more commonly of glove and stocking distribution;

(d) the paralysis may be strikingly symmetrical and maximal distally, instead of, as in poliomyelitis, asymmetrical and maximal proximally;

(e) the wasting may be less than one would expect or may not appear even after several months. This suggests that the characteristic early degenerative pathology of acute poliomyelitis is missing. Tests of the electrical reactions of the muscle, nerve stimulation and electromyography can contribute to both diagnosis and prognosis The treatment, particularly of the associated paralyses of respiration and deglutition, is of course, similar.

Acute radiculitis of the shoulder is occassionally associated with mild fever and often a good deal of pain. It may present as a case of poliomyelitis, but careful examination will show sensory loss over the outer surface of the arm and the different course of the disease usually soon differentiates the two conditions.

DISPOSAL

A practical problem for both the general practitioner and the infectious disease doctor is whether the evidence is sufficient to justify or demand admission of the case to hospital (and warning to the household contacts of their possible infectivity to others) If the case is one of acute poliomyelitis it should undoubtedly be admitted to hospital — since it is impossible in the early stages to forecast which case will develop the respiratory and swallowing complications whose prompt and efficient detection and treatment can make the difference between life and death. But does this particular case warrant the provisional diagnosis of acute poliomyelitis?

The sign whose presence or absence is probably most useful in making a decision is the meningism. A mild degree of stiff neck alone, particularly in a child, is not *by itself* sufficient evidence of a disorder of the central nervous system; but a stiff neck *and* back, or a stiff back and positive straight leg raising test, whether alone or combined with any of the other early signs of central nervous system involvement quoted above and not explained by local signs suggesting other conditions, are sound reasons for transferring a patient to a suitable fever hospital, whether the final diagnosis be acute poliomyelitis, some other condition, or no pathology proved.

In fact, approximately one-third of cases admitted to hospital with the diagnosis of acute poliomyelitis are found to be suffering from another condition, or are discharged with no diagnosis proven. This is, however, less of an error than might at first appear. Many of the alternative diagnoses require hospitalisation either for their treatment or for isolation from the community. Further, some of the cases discharged unproven probably have been cases of acute poliomyelitis, (particularly if they are known contacts), and the fact that they were fortunate enough not to develop paralysis does not detract from the wisdom of admitting them to hospital where complications could have been dealt with had they arisen.

Finally, against the inconvenience of admitting a case to hospital unnecessarily must be counterbalanced the greatly increased risk to a genuine case of delaying his admission until complications begin to develop, at which time transfer itself carries its own hazards, and every minute's delay in instituting correct treatment increases the severity of the complications.

Admission to hospital must not, of course, itself carry risk either to the patient himself or to the other patients. This is particularly important where a "polio centre" rather than a fever hospital is concerned. It is obvious that *full fever precautions* are essential to protect (a) the staff (b) the patient himself against exposure to an infection which he may, in fact, not already have acquired, and (c) to protect the other patients against whatever undiagnosed infection he may bring with him. Unless such precautions are observed not only will general practitioners be reluctant to admit cases in the early stages, (particularly reluctant if there are known to be cases of acute poliomyelitis already admitted) for fear of exposing them to poliomyelitis virus unnecessarily, but also cross-infection will sooner or later occur. If a case admitted to hospital with unconvincing signs of poliomyelitis does not develop paralysis until two or three weeks later the possibility of cross-infection by negligence of precautions should always be considered, and the possible mode of spread carefully investigated. Similarly the development of fresh cases in the contacts of a hospital dealing with the acute stage of the disease or in the contacts of its staff should stimulate a careful investigation into the security of the precautions observed there, since spread to the population in the area round a hospital treating acute cases without strict precautions has been known to occur.

CHAPTER 6

SPECIFIC TREATMENT AND PREVENTION

Specific treatment

At the time of writing there is no drug or known antibiotic which will affect the poliomyelitis viruses once they have gained access to their human host. The only agents so far known to be effective against the viruses in the body are the antibodies whose formation they themselves stimulate. The use of these antibodies is confined to prevention, and, in fact, prevention of the disease by active and passive immunisation and the control of spread of the virus are the only measures available for reducing the incidence of paralysis caused by the poliomyelitis viruses.

1. THE USE OF POLIOMYELITIS ANTIBODIES IN THE PREVENTION OF ACUTE POLIOMYELITIS

Active immunisation.

[illegible]

(non-pathogenic) strains (61, 62, 63, 64, 65, 66).

Both methods are still under trial. Both must fulfill two essential criteria:

(a) their administration must carry no risk of either an attack of acute poliomyelitis or of other undesirable complications,

(b) their antigenicity must be sufficient to stimulate antibody production against all three types to a degree indistinguishable from that following useful natural symptomless infection.

No attempt will be made to choose here between the two methods, neither of which is yet perfected. Practical considerations will presumably finally select one or other for use in particular circumstances. An essential preliminary to large-scale poliomyelitis immunisation in any community should be the provision of information on the antibody state of different ages and groups of its population and also of the incidence of the most severe forms of the disease so that the vaccines, whose production is the product

of so much careful work, *shall not be wasted in their administration* but given to those most in need of protection. At the time of writing, pregnant women, and individuals of all ages going abroad from this country to areas with high incidences of severe forms of the disease would appear to be among the most urgent candidates, *since it is they who at present provide such a high proportion of the* most severely paralysed — as well as of fatal — cases. In the U.S.A. the 5-9 year age group have been selected as the section of the population in greatest need of protection: in other areas where the incidence is highest under the age of five years active immunisation may be required before the end of the first year if it is to produce a significant reduction in the incidence of the disease.

Passive immunisation. Until active immunisation is well established passive immunisation by the administration of antibody can still play a useful part in protecting exposed individuals. It should now be possible for preparations of known antibody potency to be available for use in protection of those known to be particularly at risk. Passive immunity is, of course, only of temporary value (five to six weeks).

Three types of preparations of antibody can give passive immunity:

(i) *Individual sera* from adults or known convalescents. These, though they may contain high concentrations of any one type of *poliomyelitis antibody, cannot be relied upon to contain* useful concentrations of antibodies against all three types of the virus. Consequently their use involves both titration of each serum used and typing of the strain against which it is to be used — an impracticable proposition because of the delay it causes.

(ii) *Preparations of pooled sera:* these are more likely to be "polyvalent" than individual sera but have the disadvantage that the risk of contamination of the pool with infective hepatitis virus increases with the addition of each sample of serum; yet a large pool is desirable in order to increase the chances of representation of antibodies of the three types.

(iii) *Gamma globulin* made by concentrating the pooled sera of (ii) appears to remove the danger of hepatitis while retaining the representation of the three antibodies. The antibody content in the gamma globulin appears to be approximately the same as in the initial serum pool from which it was prepared[67]. *The larger the* pool of serum from which the gamma globulin is prepared the more

likely it is to contain useful levels of antibody against all three types of virus, provided strains of all three types are known to be prevalent in the community. It is obvious that gamma globulin prepared even from a large pool of serum collected from a population whose adults are susceptible to the prevalent virus might well be found not to contain sufficient antibodies against just that type of virus against which protection was most needed. The rational use of gamma globulin preparations demands their titration to ensure that they do in fact contain useful amounts of antibody of all three types. The levels of antibody obtained in the recipient's serum with practicable dosage of gamma globulin, which is a viscous material, are low compared with the levels which can be obtained by active immunisation, but they appear to be sufficient to neutralise virus in blood on the way to the central nervous system (though not sufficient to cross the blood-brain barrier and so to neutralise the virus already in the central nervous system ([68])). It is therefore reasonable to expect gamma globulin to have a prophylactic effect; and this effect has been confirmed by clinical trial in the United States of America where in 1951 and 1952 gamma globulin was given *prophylactically* to approximately half of 55,000 children in epidemic areas ([69]). The results suggested that the injections (a) reduced the severity of cases occurring within the first week following the injection and (b) reduced both severity and the number of cases in the following four to five weeks, being the period for which the antibody was expected to remain in the bloodstream These findings indicate that gamma globulin has a useful application in the protection of individuals particularly at risk. It should perhaps be emphasised that though, on paper, "reduction of severity of paralysis" may not appear to be a very striking achievement, in practice it may make all the difference to the patient between complete dependence and an independent, though perhaps restricted, life for the rest of his days; and in fact measures which reduce severe to moderate, or moderate to mild paralysis, are as important as measures which prevent mild paralysis

One objection raised against the use of gamma globulin to give temporary passive protection has been that its recipient might not acquire active immunity if infected.

of so much careful work, shall not be wasted in their administratio
but given to those most in need of protection. At the time o
writing, pregnant women, and individuals of all ages going abroa
from this country to areas with high incidences of severe forms o
the disease would appear to be among the most urgent candidates
since it is they who at present provide such a high proportion of th
most severely paralysed — as well as of fatal — cases. In th
U.S.A. the 5-9 year age group have been selected as the section o
the population in greatest need of protection: in other areas wher
the incidence is highest under the age of five years active immu-
nisation may be required before the end of the first year if it is to
produce a significant reduction in the incidence of the disease.

Passive immunisation. Until active immunisation is well established
passive immunisation by the administration of antibody can still
play a useful part in protecting exposed individuals. It should now
be possible for preparations of known antibody potency to be
available for use in protection of those known to be particularly
at risk. Passive immunity is, of course, only of temporary value
(five to six weeks)

Three types of preparations of antibody can give passive
immunity:

(i) *Individual sera* from adults or known convalescents. These,
though they may contain high concentrations of any one type of
poliomyelitis antibody, cannot be relied upon to contain useful
concentrations of antibodies against all three types of the virus.
Consequently their use involves both titration of each serum used
and typing of the strain against which it is to be used — an im-
practicable proposition because of the delay it causes.

(ii) *Preparations of pooled sera*: these are more likely to be "poly-
valent" than individual sera but have the disadvantage that the
risk of contamination of the pool with infective hepatitis virus
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One objection raised against the use of gamma globulin to give temporary passive protection has been that its recipient might not acquire active immunity if infected Recent studies made during the clinical trial in the United States of America, however, showed that the gamma globulin did not appear significantly to hamper the development of active immunity ([70]).

Inevitably a substance which is useful *prophylactically* is also tried *therapeutically*. In 1945 Bahlke and Perkins made such a trial of gamma globulin on a controlled series of poliomyelitis cases (71). [illegible]

barrier. Quantitative studies of the therapeutic application of gamma globulin to poliomyelitis are in fact overdue; theoretically the difficulties might be overcome by (a) obtaining levels of serum antibody sufficient to allow a spill-over into the central nervous system (29), or (b) lowering the blood-brain barrier (72) or (c) administering the antibody on the brain side of the barrier, e.g. into the cerebro-spinal fluid (73).

2. Public health measures in prevention of spread of acute poliomyelitis — the protection of the individual from exposure

As already described it has been known for many years that not only the case of poliomyelitis but also his contacts are infected with [illegible] by the time the case is diagnosed (74).

[illegible]

rare complication of a common infection, this fact of the infectivity of a case and his contacts did not appear to demand vigorous action, though in this and some other countries full fever precautions had been used in nursing the case for the first three weeks of his illness. It was generally felt that any attempt to prevent the spread of virus would be not only ineffectual in the presence of so many silent infections, but even — assuming it were possible — undesirable, since it would presumably deprive the population of harmless and potentially useful immunity.

Now, however, that the ratio of frank disease to symptomless infection has so strikingly increased — in other words that the disease has developed epidemic characters, it has become necessary to reverse this view, for the proportion of infected cases acquiring paralysis as well as immunity has become so great that the cost in death and disablement of allowing the virus free passage through the community must be regarded as excessive. A serological study of the children of a town in the United States of America illustrated this point very clearly. Even after two severe epidemics in four years (one with an incidence of 62 frank cases per 100,000 popula-

tion) over half the child population studied were found to be still susceptible to one or more types of the virus, though all three types had been represented in the epidemics ([42]).

The cost in paralysis and death of "natural immunisation" is in fact now so high that until it can be replaced by a safe artificial immunisation some form of quarantine restriction, applied not only to the case but also to the case's contacts, is necessary to obstruct the free passage of the more virulent forms of virus to other susceptible members of the community. The higher the virulence of the infecting virus (i.e. the higher the incidence of frank disease per infection) the more obviously effective will the quarantine regulations be, since many of those quarantined will develop frank disease during the period of quarantine ([45]). At lower levels of virulence the number of quarantined persons who actually develop frank disease will be less impressive but their quarantine will still have controlled the concealed spread of the virus and the same general reduction in notifications will have been achieved, particularly if the restrictions are enforced early in the poliomyelitis season: the more virulent strains of virus will have been "blind alleyed" and the chances of the population acquiring immunity without paralysis by infection with the less virulent strains of the same type will be correspondingly increased — in other words, natural *safe* immunisation without paralysis will have been favoured ([74]).

Measures recommended.

1 Of prime importance is the observance of *strict fever precautions* (aimed against both pharyngeal and faecal excretion) in handling the patient in the first three [illegible]

[illegible]

[illegible] of those coming in contact with both patients and their contacts.

2 Quarantine of contacts appears to be both an effective and practicable measure. The type of control of contacts which is used effectively in some communities for smallpox control, viz. observation and isolation when symptoms develop, is unfortunately not suitable to the control of the contacts of a case of acute poliomyelitis since an individual may be infectious for as long as 4 days before he develops frank symptoms or he may be symptomless throughout. Therefore quarantine is recommended for 3 weeks

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Inevitably a substance which is useful *prophylactically* is also tried *therapeutically*. In 1945 Bahlke and Perkins made such a trial of gamma globulin on a controlled series of poliomyelitis cases ([71]). They showed no advantage from its use. Unfortunately, the gamma globulin was given intramuscularly and the dosage used must have been inadequate to enable the antibodies to cross the blood-brain barrier. Quantitative studies of the therapeutic application of gamma globulin to poliomyelitis are in fact overdue; theoretically the difficulties might be overcome by (a) obtaining levels of serum antibody sufficient to allow a spill-over into the central nervous system ([39]), or (b) lowering the blood-brain barrier ([72]) or (c) administering the antibody on the brain side of the barrier, e g. into the cerebro-spinal fluid ([73]).

2. PUBLIC HEALTH MEASURES IN PREVENTION OF SPREAD OF ACUTE POLIOMYELITIS — THE PROTECTION OF THE INDIVIDUAL FROM EXPOSURE

As already described it has been known for many years that not only the case of poliomyelitis but also his contacts are infected with *and excreting poliomyelitis virus by the time the case is diagnosed*([74]). So long as infection with the poliomyelitis viruses was in the vast majority of individuals symptomless, and frank disease only a rare complication of a common infection, this fact of the infectivity of a case and his contacts did not appear to demand vigorous action, though in this and some other countries full fever precautions had been used in nursing the case for the first three weeks of his illness. It was generally felt that any attempt to prevent the spread of virus would be not only ineffectual in the presence of so many silent infections, but even — assuming it were possible — undesirable, since it would presumably deprive the population of harmless and potentially useful immunity.

Now, however, that the ratio of frank disease to symptomless infection has so strikingly increased — in other words that the disease has developed epidemic characters, it has become necessary to reverse this view, for the proportion of infected cases acquiring paralysis as well as immunity has become so great that the cost in death and disablement of allowing the virus free passage through the community must be regarded as excessive. A serological study of the children of a town in the United States of America illustrated this point very clearly. Even after two severe epidemics in four years (one with an incidence of 62 frank cases per 100,000 popula-

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Measures recommended.

1 Of prime importance is the observance of *strict fever precautions* (aimed against both pharyngeal and faecal excretion) in handling the patient in the first three weeks of the disease These must be a *sine que non* of the management of poliomyelitis, the infectivity of the pharynx as well as of the faeces should be uppermost in the minds of those coming in contact with both patients and their contacts.

2 Quarantine of contacts appears to be both an effective and practicable measure The type of control of contacts which is used effectively in some communities for smallpox control, viz. observation and isolation when symptoms develop, is unfortunately not suitable to the control of the contacts of a case of acute poliomyelitis since an individual may be infectious for as long as 4 days before he develops frank symptoms or he may be symptomless throughout. Therefore quarantine is recommended for 3 weeks

from the time of contact for household or other known direct contacts, by the Expert Committee of the World Health Organisation and the Ministry of Health for England and Wales [76]. On occasions it may be necessary to control second-class contacts (contacts of contacts) such as the family or household of a contact, particularly if these have special opportunities for transmitting the virus to large numbers of others, e.g. school teachers and school children. *The 3-weeks period, though it is of course shorter than* the period of faecal infection, appears to cover the period of infectivity. The combination of fever precautions in nursing a case, quarantine restrictions of his contacts, and the administration of gamma globulin to contacts likely to be particularly susceptible (the pregnant, the lactating and those within one month of an irritating injection) offers a practical method of controlling the disease, until active immunisation becomes an effective solution to the problem.

CHAPTER 7

MANAGEMENT OF THE ACUTE STAGE

Since there is no specific therapeutic agent yet available for the control of the infection once clinical disease has developed, treatment is largely palliative and directed to the prevention and control of complications. It is useful to bear in mind the following characteristics of a patient with acute poliomyelitis:

(1) *He is acutely infectious* to all those who come in contact with him or his excreta, and for the sake of his attendants and of all those who come in contact with them *full fever precautions* must be taken as if he were a case of both diphtheria (infection in the pharynx) and typhoid (infection in the stools). Masks and gowns must be worn, and all excreta and linen disposed of so that transmission of the virus cannot take place.

(2) [illegible]

traction on the cord and nerve roots He may tend to adopt, so far as his muscle power allows, a version of the characteristic "meningitic" posture and to maintain it by "spasm" All this motor activity, whether reflex or voluntary, is however, believed to contribute to the vulnerability of the nerve cells to the virus; it is therefore as important as it is humane to reduce it by dulling his consciousness and encouraging sleep. A striking feature of the disease is that pain and surrounding events may be appreciated very intensely, even though the interpretation of them may be somewhat confused

(3) *He is in part or wholly at the mercy of his environment* as a result of the paralysis of the muscles of head, neck, trunk and limbs It is often hard to appreciate what this means to the patient in the acute stage when his limbs look quite normal.

(4) *Respiration and swallowing, micturition and defaecation may be paralysed* as a result of damage to the innervation of their muscles or to the vital centres. Paralysis of respiration and swallowing may develop extremely rapidly and are an acute medical emergency in which the prognosis deteriorates with every minute's delay in treatment. It is therefore essential that a close watch be kept for the

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indication that the attack of poliomyelitis is particularly severe or his life in danger. He should also be reassured that its use will be not a further infliction in his plight, but a pleasant relief which in particular will allow him to get much-needed sleep.

2. Relief of pain

Pain should always be relieved as far as possible in order to reduce motor activity, restlessness and spasm:

(a) *Aspirin* relieves the headache, reduces the fever and consequently the discomfort, and also has a useful analgesic action.

(b) *Phenobarbitone* is almost specific in relieving the often acute awareness, discomfort, and anxiety. Doses of gr. 1 t.d.s. for adults are recommended and a proportionate dose for children. A patient *in hospital* and therefore within reach of all accessory life-saving [illegible]

recovering their normal general health, cannot resume their normal activities. Its depressant effect on morale should not, however, be forgotten.

Opiates are, generally speaking, contraindicated because they depress the respiration and the cough reflex and because they may cause vomiting, which is particularly undesirable since the vomit may be aspirated. Small doses of nepenthe may, however, be useful occasionally, when possible complications are all under control (i.e. the patient is already adequately ventilated in a lung and all equipment for suction is available), if there is particular need of it for some unpleasant or tiresome manoeuvre. Thus for prone turning in a respirator the minimum of nepenthe coupled with a short acting barbiturate is permissible and helpful; but after its use a very careful watch must be kept for signs of underventilation or obstruction of the airway

(c) Finally, it should be stressed that judicious measures to relieve spasm, the correct posturing of the limbs, and minor shifts of weight to relieve pressure as requested by the patient are essential parts of the relief of pain; and that good nursing is more effective than the entire hospital pharmacy in relieving the patient's discomfort, pain and anxiety, and in preventing severe complications

earliest signs of their development throughout the first two weeks of the disease.

Inevitably, the nursing of acute poliomyelitis is a compromise. We should like to abolish all motor activity by heavy sedation but we must keep the patient sufficiently awake for him to assist voluntarily in the correction of paralyses of respiration and swallowing

to sleep with an effective drug before we have taken over from him these two important tasks he will either drift via the unconsciousness of sleep through anoxia into coma and death, or simply drown without a struggle in his own saliva.

Similarly, we should like to disturb him as little as possible, in order to reduce the reflex motor activity caused by handling him. But if we carry out this principle without modification, he will, if he is paralysed, develop deformities and pressure sores; if he has respiratory weakness he may develop hypostatic pneumonia and atelectasis, and if his recumbency is prolonged, he will run the risk of urinary complications.

1. Reassurance

An atmosphere of optimism should prevail. It is impossible, even for the most experienced clinician, to forecast in the early days what will be the final disability, and nurses as well as patients should be assured that even severe initial paralyses may show very reasonable recovery. Bulbar cases, in particular, often have an excellent prognosis if they can be helped to survive the complications of the acute stage.

The patient should be told that his most important contribution is to relax, that he will be helped to do so by drugs, and that the protection and care of his limbs will be taken over from him. This promise must, of course, be fulfilled. He must be instructed to refrain from "trying muscles to see if they work" and from trying to "work the numbness out of them." If assisted respiration is likely to be necessary he should be told *beforehand* that the "iron lung" will be used as a temporary assistance — to take over from muscles before they become exhausted — and that its use is not an

indication that the attack of poliomyelitis is particularly severe or his life in danger. He should also be reassured that its use will be not a further infliction in his plight, but a pleasant relief which in particular will allow him to get much-needed sleep.

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[illegible]

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convalescent stage, to relieve the frustration of patients who, while recovering their normal general health, cannot resume their normal activities Its depressant effect on morale should not, however, be forgotten.

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3. Correct management of paralysed parts

(a) *Maintenance of normal or optimum posture.* The patient lies flat on his back, *(fig. 8) the head is supported on a flat pillow,* and the normal lordosis is maintained supported by a very small pillow under the lumbar spine.

The ideal position of the paralysed arm is abduction at the shoulder, moderate flexion of the elbow and the position of function of *the hand (dorsi-flexion of the wrist, opposition of the thumb and* slight flexion of the fingers) (fig. 9). The purpose of the abduction at the shoulder is to maintain the length of the anterior and posterior axillary walls and not, as is often believed, to "rest" the deltoid.

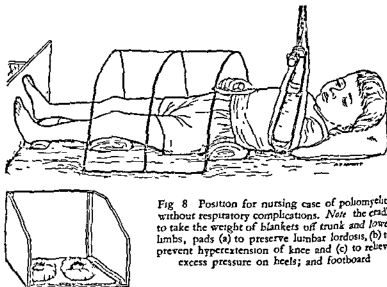
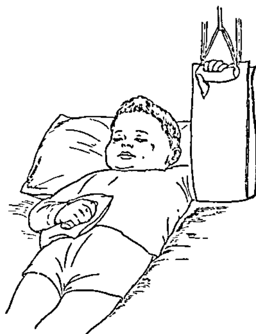


Fig 8 Position for nursing case of poliomyelitis without respiratory complications. *Note* the cradle to take the weight of blankets off trunk and lower limbs, pads (a) to preserve lumbar lordosis, (b) to prevent hyperextension of knee and (c) to relieve excess pressure on heels; and footboard

In most current types of respirators it is not possible to obtain satisfactory abduction at the shoulder, and it is necessary to have the arms either straight at the side or supported in as much abduction as possible with the forearm flexed on a small pillow, so adjusted that it does not interfere with the chest movement. *In the* new wider respirators some abduction at the shoulder is possible and should always be taken advantage of; since adduction contracture of the shoulder is, because of the difficulty of fixing the scapula, a difficult complication to correct later. It may take many months

to overcome such a contracture which is too often fully developed after four weeks of respirator treatment.

For the lower limbs slight flexion of hip and knee are the position



which even a normal leg becomes uncomfortable most quickly.) Lateral support at knee and foot is usually also required to prevent the limbs rolling into external rotation.

The foot is maintained in the neutral position — no dorsi-flexion, no adduction, no inversion or eversion — by any method which is successful. It is important to prevent shortening of the calf

muscles. A simple foot-board is often sufficient in a completely paralysed patient, but if the patient can move either his trunk or the limb, foot and footboard will soon part company. The hasty readjustment by nurse or physiotherapist every time the bedclothes are turned back (often with earnest protestations that the position was excellent half an hour ago) is an indication that the device in use is inadequate for this particular case. A useful device is the half-box (fig. 8) with a heel ring to distribute the weight of the heel and keep the foot from slipping away from the end; its sides control the tendency of the foot to rotate outwards; in the presence of a flail hip, however, this control must be supplemented [illegible]

plaster or aluminium back splint: it must of course extend above the knee, since the calf muscle covers both knee and ankle joints; if only a below-knee splint is used the calf will shorten by flexion of the knee, and when the splint is removed full extension of the knee is obtained only at the expense of dorsi-flexion of the foot, and vice versa.

In the prone or, better, the semi-prone position the head is turned to the side; the chest and pelvis are supported on pillows to allow movement of the diaphragm and abdominal muscles and the legs must be supported at the ankle to allow the feet to drop into the neutral position. Abduction of the arms to 90° with flexed elbows has the advantage of correcting the tendency to adduction contracture but it is often too painful to be tolerable in this position.

(b) *Careful handling of the limbs.* As indicated above, the utmost care is necessary in handling the limbs in a case of acute poliomyelitis. Nurses should be taught to

(i) avoid stretching the nerve roots, which precipitates or increases spasm;

(ii) avoid compressing the muscles. The limbs should always be picked up at at least two bony places, and never over a muscle belly, which is always sensitive — particularly sensitive is the calf, which is also particularly exposed to handling. The lower limb should be picked up by one hand under the os calcis and the other under the knee, and not by the foot alone since this allows the

weight of the leg to hyperextend the knee, and at the same time inadvertently performs a straight leg raising test, which we know to be painful.

(iii) avoid distortion of the unprotected joints. Subluxation of the hip and shoulder has been known to occur particularly in small children unable to express or resist the pain caused by distorting movements. This danger should always be in the mind of a physiotherapist bent on maintaining full range of movement.

(c) *The prevention of postural oedema.* Gross oedema of the feet and legs is occasionally seen when there is complete paralysis of the lower limbs. The immobility of the legs in a dependent position, and compression of veins and lymphatics in the popliteal region and upper calf are the causes; passive movements, massage and correct posture, combined where necessary with a slight head-down tilt of the bed or respirator at night prevent it.

(d) *Protection against pressure sores.* The mattress should be flat; if necessary, fracture boards should be applied under the mattress which should be at least 5 inches thick (for an adult). Either interior springing or a foam rubber mattress is the most suitable foundation for a paralysed patient.

The weight of bedclothes must be reduced to a minimum since by increasing the pressure of the patient on the bed, they increase the tendency for sores to develop. In addition, they cause deformity — particularly of the feet — and can seriously interfere with respiration. The weight of the bedclothes sh[illegible]
cra[illegible]
bla[illegible]
massage with spirit must be a routine, and pressure rings (pneumatic or the usual bound bandage ring) by dispersing the weight contribute to the prevention of sores over pressure points. Attention must also be paid to any site which may be compressed by apparatus, e.g. the exposed position of the external popliteal nerve to pressure by a plaster back-slab, and the shoulders of a patient who is tipped head downwards in a cabinet respirator.

(e) *Maintenance of passive joint range and the relief of spasm.* The maintenance of a useful range of joint movements is important in the early stage of the disease, for, as already indicated, it is at this stage, when denervation changes are taking place in the muscles

that the tendency to develop either shortening or excess length of *the affected muscles is greatest. The need to maintain a useful* range of movement, however, in no way contradicts what has already [illegible]
limbs. [illegible]
moveme[illegible]
produces, not only is agonisingly painful for the patient, but also defeats its own object, since it only increases the spasm to be contended with at the next session; in children it can lead to dislocation, particularly at shoulder or hip.

Maintenance of a *full* range of movement is, in fact, in many cases not possible; rather a "useful" range of movement should be aimed at. Thus, if passive abduction of the arm to 90° can be maintained in the acute stage, there will be little difficulty in restoring *full range in the transitional stage. (In certain joints, in fact, full* range of movement is not even desirable — e.g. full palmar-flexion of the wrist with weak dorsiflexors or full dorsiflexion of the foot with a paralysed calf can be a severe disability)

Passive movements are performed so as to give as little pain and fatigue to the patient as possible. If movement at a joint is painless and no resistance is felt it need not be put through its range of movement more than once — to confirm that the movement is in fact free. If there is resistance — or pain — this is best overcome by taking the joint gently to the limit of the patient's comfort (*not of what can be obtained by force*) *reversing and then approaching* the limit again — at each return a small increase in range will be obtained without any increase in pain. "Spasm" of the antagonists is abolished not by force but (a) by asking the patient to assist the movement gently, since in attempting to do so he will inhibit the unconscious resistance of the antagonists and (b) by the application of hot moist packs to the muscles and their antagonists before performing the movements.

Kenny treatment

Treatment by hot packs was introduced into the treatment of acute poliomyelitis by Sister Kenny of Australia (73 79). It consists in the application of heat in the form of hot fomentations over muscles whose shortening, whether of denervation or of spasm, is painful and restricts the range of joint movement. The choice of *moist* heat is necessary because other forms of external

considerable help in the control of painful spasm and maintenance of passive movement in the acute and transitional stages. If there is

pain, and the help they give in putting joints through their necessary range of movement. The hot packs are applied in the form of pieces of blanketing wrung dry through boiling water and applied direct to the skin The moisture is retained with jaconet or other impermeable material and the heat by further warm wrapping outside the jaconet. Sister Kenny was insistent that the pack should not cover the joint itself but be applied to the different units of the body leaving the joint free, and this is of course the most practicable way to apply them. A special machine which spin-dries has been designed for the preparation of hot packs but a simple washing machine, with attached wringer, through which the packs are wrung twice, is perfectly suitable for the occasional patient In hot weather the use of packs may have to be restricted because of the danger and unpleasantness of overheating the patient, particularly if he is febrile. Since hot packs owe their action to their heat, which causes a reflex relaxation of muscle, their effect is reversed when they become cold; it is therefore obvious that they should *never be left on for longer than useful heat is retained.* A useful routine for the application of packs in the acute stage is a two-hourly administration, the packs being applied five times a day, at two-hour intervals If the interval is to be longer, as at the end of the day, they are removed at the end of two hours. They must not of course be left on over-night, nor should their application be continued through the night since due respect must be paid to the patient's need for sleep. In practice, packs applied through the day will usually achieve sufficient comfort for the patient to pass the night without further attention. In this context, it should be mentioned that there is always a danger of excessive enthusiasm leading to over-treatment of acute poliomyelitis. Poliomyelitis patients, particularly children, whose need for sleep is always high, must not be handled and manipulated continually but allowed to rest, and treatment and interference should always be cut down to the minimum compatible with the needs of the

respiratory system, and of the paralysed limbs, and adapted to the pa[illegible]

ho[illegible] again, their effect is probably by relieving reflex spasm. Repeated short applicatic[illegible] three minutes, [illegible] considerable help when respiration is impaired and may in the borderline case obviate the need for artificial respiration. A patient who can breathe in the supine position but becomes breathless in the prone position can sometimes be considerably relieved in the prone position by such intensive packing to the back.

A further use of hot packs may be mentioned here. It arises after the acute stage when the patient is seen with already established contractures, e.g. a patient coming out of a cabinet respirator with adduction contractures of the shoulders. In these cases hot packs can help to relax and stretch the already established contractures At this stage it is often found best to concentrate the packing in only one or two single sessions a day, during which the packs are changed every 10 minutes with the aim of maintaining a high temperature for about one hour, at the end of which time gentle, but firm, manipulations of the affected muscles and joints may be attempted with good chance of success. If the limb is now left in the position of maximum stretch of the contracture the next treatment starts at this point, and a few further degrees of stretching are obtained daily with minimal pain.

4. Management of urinary difficulties

Paralysis of the bladder with retention of urine is a relatively common complication and should be watched for and treated as soon as it develops. Normal function usually returns within two weeks. Treatment may be either by the administration of carbacol or similar drugs, or by catheterisation twice a day with all sterile precautions, never by a self-retaining catheter, which increases the risk of urinary infection — a complication it is important to avoid in any paralytic condition. The period of catheterisation should be covered by antibiotics. Normal bladder function usually returns within two weeks. Prolonged retention, or its recurrence after the acute stage is usually associated with neglected constipation and responds to effective clearance of rectum and colon.

5. MANAGEMENT OF CONSTIPATION

Constipation is a very common complication. While it lasts, it can, if neglected, cause a considerable degree of discomfort, is often responsible for persistent unexplained mild pyrexia in both children and adults, in respiratory cases can seriously restrict the [illegible]
c [illegible]
t [illegible]
of passage of small stools — or even of unexplained faecal incontinence and the "diarrhoea" of paralytic constipation. In such cases effective evacuation of the bowel with glycerine enemata, or, if necessary, manually, will produce the most astonishingly bulky results. This will be followed by marked improvement of the general condition of the patient and, if the respiratory muscles are affected, also of the respiration. Constipation is, of course, most often seen in the severely paralysed who are inactive, and particularly in those with weakness or complete paralysis of the abdominal wall. So long as it persists it must be controlled. The following routine regime is recommended:

A glycerine enema is given every third day (2 oz. equal parts glycerine and water, for children, and 4 oz. equal parts glycerine and water for adults). Once the bowel has been effectively evacuated the constipation can usually be controlled by small amounts of liquid paraffin given by mouth or by any aperient found to suit the patient, and only occasional enemata will be necessary. The tendency to constipation decreases with increased activity.

6. DIET

In the early stages diet should be nourishing but light Digestibility and rapid passage through the stomach is an important factor, since vomiting is common, particularly in those with incipient bulbar paralysis. In the acute stage all feeding should, if possible, be done by the nurse in order both to rest the patient and to keep his swallowing under observation [illegible]

[illegible]

difficulty in swallowing can often control "jelly" better than fluids (milk or water jelly) and finds particular difficulty with foods of more than one consistency, e.g. fruit salad, cornflakes with milk, etc. or very hard foods

After the acute stage the diet must be adjusted to the needs of the patient. Nearly all patients lose a good deal of weight during the acute stage and they should at first have a high protein diet and be allowed and encouraged to eat as much as they like. After the first three or four months, however, a watch should be kept on their weight so that they do not overburden weakened muscles with an unnecessary load of fat. Cases who have had respiratory complications are usually slow to put on weight and will be unaffected by every fattening ruse. There seem to be two reasons for this — firstly a patient with significant respiratory deficiency takes what seems to his attendants an incredibly long time to eat even a small meal, and his intake is consequently reduced; secondly, a full stomach increases the patient's difficulty in breathing and he therefore avoids it.

CHAPTER 8

RESPIRATORY COMPLICATIONS

In the term "respiratory complications" we include all the complications arising from paralysis of the muscles whose normal function is to keep a clear airway and maintain adequate ventilation of the lungs: viz. the muscles of the tongue, jaw, palate, pharynx and larynx on the one hand, and the true "respiratory muscles" (diaphragm, intercostals and abdominal muscles) and accessory muscles of respiration (sternomastoid) on the other. Paralyses of these muscles constitute an acute medical emergency; they are responsible for the majority of deaths occurring in the acute and later stages of the disease. Correct *early* management of the complications is important in the reduction of the mortality rate of the disease.

Clinically the respiratory complications of acute poliomeylitis fall into three categories:

1. *"Bulbar" cases* — in which the complications are caused primarily by paralysis of the muscles of the tongue, jaw, palate, pharynx and larynx — with a resulting inability to keep the airway patent and free from vomit, saliva, food, etc.
2. *"Spinal" cases* — in which the complications are caused primarily by paralysis of the muscles of respiration — with a resulting inability to maintain sufficient movement of the lungs.
3. *"Bulbo-spinal"* cases — in which the two first categories are combined

AUTOPSY FINDINGS IN DEATH FROM RESPIRATORY COMPLICATION

The autopsy findings are similar for all three types. In cases which die early in the acute stage, or suddenly from an acute episode, the main finding is widespread pulmonary oedema. The [illegible]

[illegible]

In cases which die later, or more slowly, atelectasis is the more common finding Often there is free bronchial secretion, and, if

infection is present, varying degrees of bronchitis, with lobar or scattered pulmonary collapse or early pneumonitis. Sometimes, however, the lungs are simply dry and airless.

The fluid which appears in the lungs may have either or both of two main sources:

(i) It may have been suddenly aspirated from the pharynx in the form of saliva, drink or vomit, or have slowly trickled down over the course of many hours.

(ii) It may have developed as a pulmonary transudate secondary to the defective ventilation. Transudation from the capillaries can occur both from the direct effect of anoxia on their permeability and indirectly as a result of the raised capillary pressure caused by the rise in the systemic and pulmonary blood pressures associated with both anoxia and hypercapnia. Transudation is also more likely if excessive amounts of intravenous fluid are given or if the serum albumen falls.

The presence of fluid in the airway still further decreases the respiratory exchange with the result that the patient enters a descending spiral of defective oxygenation, pulmonary transudate and still further interference with the respiratory exchange, which, if not detected and treated, will lead to death.

The biochemical changes accompanying impaired ventilation depend on the rate with which the deficiency develops. If the accumulation of carbon dioxide is rapid the resulting respiratory [illegible] the blood pH falls. If the accumula-

[illegible]

which in this instance is best regarded as the amount of bicarbonate required to buffer the carbon dioxide, rises.

THE PREVENTION AND CONTROL OF RESPIRATORY COMPLICATIONS

The prevention and control of respiratory complications resolves into two essentials:

(1) the airway must be kept clear from mouth to alveoli and

(2) the lungs must be moved sufficiently to ensure adequate respiratory exchange.

1. THE MAINTENANCE OF A CLEAR AIRWAY.

It is obvious that obstruction of the airway causes poor ventilation, *however large the force applied to the lungs.*

The patient with poliomyelitis gets an obstructed airway *either* because he cannot shut off his larynx and swallow to clear his pharynx *or* because he cannot cough up the material entering or being secreted in his lungs. His difficulties are increased by the adverse slope of the respiratory tract in the supine position:

The 15° slope of the respiratory tract (see fig. 10).

In the supine position (the usual position for a patient) there is a 15° downhill slope from the mouth to the hilum. This means that there is a constant gravitational tendency for anything in the mouth, be it saliva, vomit, food or drink, to run down the trachea into the lungs and for pulmonary secretions to stay in the most dependent part of the lungs.

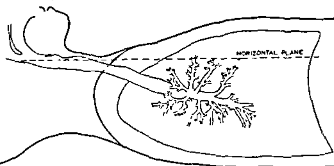


Fig 10 The 15° slope of the respiratory tract

The normal individual without paralysis does not suffer from this anatomical arrangement because (a) his muscles of deglutition remove foreign material and saliva from the pharynx and shut off the larynx, so preventing fluid from running down or being aspirated into it, (b) any large amounts of material which should enter the tract from above or be secreted into it below can be forced towards the pharynx by coughing. Smaller amounts of pulmonary transudate and secretions are simply passed up the bronchial tree by the ciliary action of the epithelium and swallowed automatically

Obstruction of the airway in poliomyelitis occurs.

(a) in the *pharynx*· when swallowing is impaired saliva, vomit, food, drink and pulmonary secretions pool at the back of the pharynx, to be inhaled sooner or later unless removed (1½ litres of saliva may be secreted by an adult in 24 hours) (Fig. 11).

(b) in the *larynx*: loss of control of the muscles of the pharynx and larynx and loss of coughing power allow the larynx to be blocked by material entering from the pharynx above, or by copious or thick secretions coming up from the respiratory tract below. Rarely, obstruction occurs from imbalance of the intrinsic muscles causing narrowing of the lumen. Spasm of the adductors or paralysis of the abductors of the larynx preventing temporary passing of an intratracheal tube is the only — and rare — indication for an emergency tracheotomy in poliomyelitis.

(c) in the *trachea and bronchi*: loss of coughing power and hypostasis from impaired respiratory movement allow material coming down from above or pulmonary secretions coming up from below to block trachea and bronchi. Areas of atelectasis develop varying from small patches to one or more lobes, according to the size of bronchus which is blocked.

It is obvious that when fluid collects in the airway (a) the adverse slope of the trachea must be overcome and (b) suction must be used to remove the fluid. It is vital that all should know that when a choking fit occurs or secretions develop the patient *should not be sat up* but should at once be tipped steeply head down. A child if necessary may be held up by the heels.

Reversal of the 15° slope into the lungs

In *the steep head-down supine position*, secretions flow down the trachea away from the lungs but tend to form a pool in the back of the pharynx where an efficient water seal can develop when the epiglottis is reached. Constant vigilance and frequent use of the sucker is necessary, therefore, in the supine position, if the airway is to be kept clear. (If this position is maintained for long periods the upper lobes may sometimes fill with fluid.)

In *the head-down prone position* secretions and saliva flow not only into the pharynx but on into — and out of — the mouth. Since the tracheal slope is reversed, only 5-10 degrees of tilt are necessary, but obstruction can still arise, if the neck is extended leaving little room for a clear airway, and from pooling in the laryngeal prominence The opening of the larynx it must be realised is now *underneath* the opening of the oesophagus and so particularly exposed to blocking by vomit.

These dangers can be partly removed by turning the head laterally or better by using the *head-down semi-prone position*. In this position,

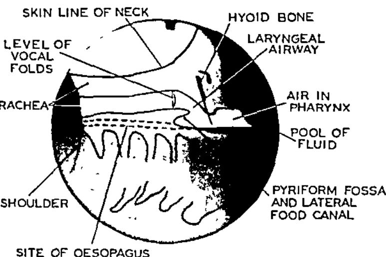


Fig 11 Radiograph with outline drawing of pooling in the back of the pharynx in a child with paralysis of the muscles of deglutition. The importance of adequate suction of the pharynx in these cases *before* turning the patient on to his face is obvious.

(By kind permission of Drs. F H. Kemp and G M Ardran of the Nuffield Institute, Oxford.)

with the head turned laterally, no pools form in either larynx or pharynx and the fluid collects in the cheek, whence it either runs out of the corner of the mouth or is easily removed by suction. The semi-prone position is more comfortable since the weight is not on the sternum and there is more room for movement of one side of the chest and the diaphragm. In addition the patient can be changed from one side to the other. Unfortunately, in the face down position, respiratory movement is restricted by the weight of the trunk and mechanical respiratory aid will be required for a lesser degree of associated respiratory paralysis. In cases with slight respiratory paralysis as well as paralysis of swallowing one may have to choose between nursing in the steep head-down supine position without respiratory aid, or the less steep prone position with aid.

2. Ventilation of the lungs

Inspiration is normally performed by the intercostals (T 1-12) which expand the chest wall upwards, forwards and outwards, and by the diaphragm (C 2-4) which expands it downwards and forwards. The sternomastoids (Cranial 11) may act as accessory muscles of inspiration when ventilation is impaired; they are often the only apparently active inspiratory muscles in spinal poliomyelitis, since the other accessory muscles are so often paralysed with the rest of the shoulder girdle. Unlike the true respiratory muscles the accessory muscles do not appear to act in deep sleep.

Expiration is normally performed by the elastic recoil of the lungs and chest-wall and by the effect of gravity on the diaphragm in the horizontal supine position it can be assisted by the action of the internal intercostals (T 1-12), the abdominals (T 8-12) which, by increasing the abdominal pressure, push up the diaphragm, and the latissimi dorsi (C 7)

Coughing. Both inspiratory and expiratory muscles are required for an effective cough (effective inspiration in order to obtain sufficient volume of air; effective expiration to force out the inspired air at sufficient pressure to clear an obstruction or move secretions towards the outlet). Additional requirements are: an active cough centre to initiate the mechanism and active muscles of the larynx to allow pressure to be built up in the lungs and suddenly released.

Control of volume, rhythm and rate of respiration. The respiratory centre in the medullary bulb controls the respiration in response to

the carbon dioxide oxygen and H-ion contents of the blood.

In poliomyelitis certain unusual repiratory movements are occasionally seen and deserve mention:

(a) The sternomastoids are working alone; in this case inspiration is achieved entirely by lifting the chest wall forwards and upwards; ventilation is maximal in the upper lobes and the sternocostal angle may diminish and the diaphragm be pulled up in inspiration. Expiration is by elastic recoil. The action of the sternomastoids is grossly interfered with if the neck is flexed or the head left unsupported. Such patients cannot tolerate the prone position without mechanical respiratory help

(b) The diaphragm is working alone; on inspiration the abdominal contents are thrust downwards and forwards (the forward thrust is explained by the more caudal attachment of the posterior part); the upper ribs tend to be sucked in (paradoxical respiration) and the larynx to be pulled downwards (tracheal tug). Expiration *is by active contraction of the abdominal muscles, if they* are working, and — in the flat or head-down supine position — by the weight of the abdominal contents tending to push up the diaphragm. Ventilation may be impaired when the patient is turned on his face — in which position the diaphragm needs to act against the weight of the trunk; and when he is sat up to more than 30-40°, in which position the weight of the abdominal contents no longer pushes the diaphragm back. If the abdominal muscles are paralysed the diaphragm comes to rest in the expiratory position.

EQUIPMENT FOR POSTURAL DRAINAGE AND SUCTION WITHOUT ARTIFICIAL VENTILATION

(1) *The bed.* A head-down tilt to an angle of at least 25° is required. If neither a special bed nor an elevator is available the feet (not the [illegible] raised on chairs — or- [illegible] a *25° slope;* [illegible] so obtained is not lost by supporting the head and shoulders on pillows. A pelvic sling attached to the foot end of the bed may be necessary to [illegible] of the bed is [illegible] *of the bed.* A *cardiac bed with the patient's* head at the foot end and his pelvis raised on the "knee" angle can also be useful — though inelegant.

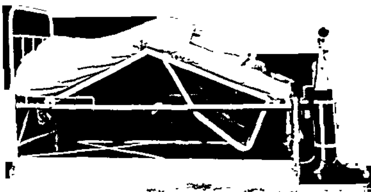


Fig. 12. Postural bed for (i) Management of patient with paralysis of swallowing. (ii) Drainage of chest in patients with inadequate cough
Note the removal of head of the bed to give ready access to the patient's face: oxygen cylinder with suction attachment
The bed can also be tilted laterally. In the absence of special equipment *either* simple tilting of the bed on chairs or on an elevator *or* the knee bend of a Fowler bed can be used

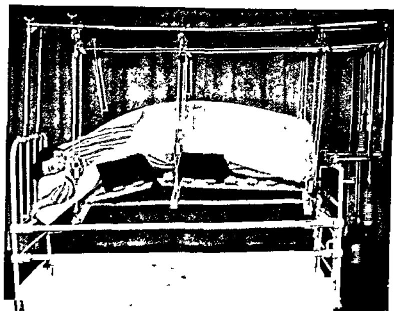


Fig 12a. Postural drainage in a suspended bed, note sideways tilting as well
(Reproduced by courtesy of the Institute of Orthopaedics.)

Special beds for postural drainage can considerably reduce [illegible]

and the column of venous pressure from feet to head is reduced, (b) capable of simultaneous lateral tilting (fig. 12). Another device is the bed board similarly articulated at the level of the patient's hips, and then slung by six pulley systems from a double Balkan beam, an arrangement which enables a wide range of tipping angles to be obtained with little disturbance to the patient (fig. 12a). It is important, in all these systems, that the security of patient, mattress and bed be each and all ensured; a completely paralysed patient will slide where an unparalysed subject will remain stable without conscious effort.

(ii) *Suction apparatus.* There are three main types of suction apparatus: viz *the electric pump*, (seen in fig. 17), *the gas venturi pump* (seen in fig. 12) and *the water pump*. All are suitable. If a water pump is used it is important to prevent contamination of the drain by introduction of an efficient trap for infected material in the system. The catheters used should be reasonably soft but at the same time sufficiently rigid for the end to be controlled from a distance and to prevent their collapse the moment any resistance is encountered. They should have no side holes (which reduce the effective suction) and should be cut off square at the end with one or two nicks in the rim, so that they will not suck onto the mucosa. For bronchial toilet through a tracheostome a catheter with a curved tip (e.g. Tenmann's) which enables the user to direct the end into either right or left bronchus may be preferred. A glass connection should always be inserted into the suction system near the patient so that the operator can easily see the material aspirated. The use of the sucker should be as gentle and atraumatic as possible.

(iii) *The throat microphone* ([79]). This consists of (a) a microphone, which can be laid on the larynx of the patient, (b) an amplifier and (c) a loud-speaker ([80]). It is a useful but not an essential part of equipment. It assists early detection of high airway obstruction. If the airway is unobstructed quiet breathing is heard as fluid accumulates in the pharynx or larynx loud clicking sounds occur, and the nearer the site of the accumulation lies towards the larynx and the trachea, the more snoring and rhonchus-like become the sounds. The microphone can be left on continuously, if the airway is clear it will not annoy the ward, if secretions are accumulating it

gives earlier warning than unaided clinical observation. It also enables a nearly voiceless patient, who cannot use a bell, to call a nurse.

EQUIPMENT FOR ARTIFICIAL VENTILATION OF THE LUNGS

All mechanical aids to respiration in poliomyelitis act by producing regular changes in the pressure inside or outside the lungs so that air — or other gases — move from the area of high pressure to the area of lower pressure. The reverse movement is usually obtained as a result of elastic recoil of the tissues when the force causing the change in pressure is removed.

The various devices for artificial respiration in common use are:

Type A. Devices in which negative pressure is applied round the trunk, to cause inspiration and expiration is by elastic recoil or by a smaller degree of positive pressure (tank and cuirass respirators).

Type B. Devices in which positive pressure is applied round the trunk to cause expiration and inspiration is by elastic recoil (the Bragg-Paul respirator).

Type C. Devices in which positive pressure is applied to the airway via a face mask, mouthpiece or tracheotomy tube, and expiration is by elastic recoil or by negative pressure (resuscitators, and intermittent positive pressure respirators for use with intratracheal intubation).

Type D. Devices which use gravity to move the diaphragm (the rocking bed).

Type E. Devices which stimulate the muscles themselves to perform the respiration (electronic muscles stimulators).

TYPE A. DEVICES.

(a) *The cabinet type* (see figs. 13 and 14). This is the only satisfactory respirator in the acute stage. In the modern cabinet respirator most of the disadvantages of earlier models have now largely been overcome, so that it is now possible to combine adequate ventilation with adequate postural drainage, even in the presence of total paralysis. In a good cabinet respirator:

(i) Ventilation is adequate in the presence of total paralysis of respiration.

(ii) Placing the patient in the respirator is simple, and little or no readjustment of the seal around the neck is necessary at each opening

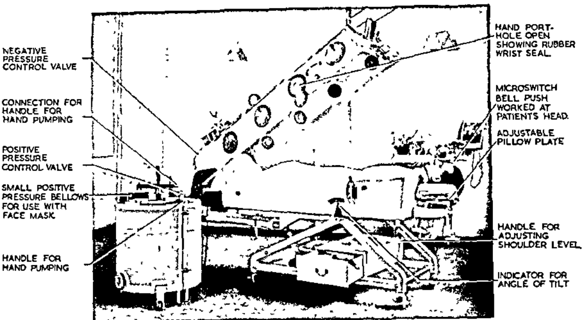


Fig 13 'Alligator-type' cabinet respirator, shown open.
(By courtesy of Cape Engineering Co, Warwick.)

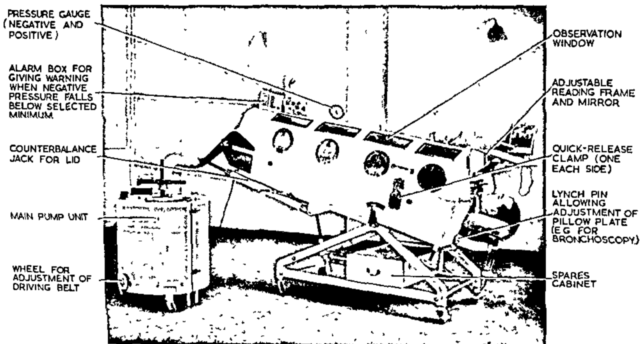


Fig. 14 'Alligator-type' cabinet respirator, shown closed and tilted for drainage from pharynx and chest. Note that the respirator can be opened while still tilted

(iii) The patient can be easily tipped head-down to as much as 25° for drainage of the air passages and for the prevention of aspiration of material from the mouth and pharynx.

(iv) The head piece is adjustable so that bronchoscopy can be performed without moving the patient from the respirator.

(v) The patient can be turned on his face.

(vi) A (high) tracheotomy can be used if necessary. (A bent strip of metal can be used to keep the collar away from the tube)

It should perhaps be mentioned that even the older respirators which do not fulfill all these requirements have saved, and continue to save, many lives and should not be regarded as useless in the absence of the more modern features, although these have added considerably to the ease and comfort of management of these cases. It remains true that an experienced and ingenious team using an old-type respirator and supported by a skilled bronchoscopist to assist in removal of major obstructions in the bronchial tree, can often achieve as much as (or more than) the more modern devices in less experienced hands. If the angle of tilt provided is insufficient and prone turning impossible effective drainage can be obtained by tilting the whole respirator on blocks, alternately laterally and head-down, using a pelvic sling to help steady the patient.

(b) *Cuirass respirators* (see fig. 16). These give less efficient ventilation than the tank respirator and are inadequate in the presence of complete paralysis. This is largely because the forces are exerted on a much smaller area of trunk, and movement at the bases tends to be restricted by the pressure of the seal on the chest wall. Cuirass respirators are useful (a) for transport (of the incompletely paralysed patient) and (b) after the acute stage, when access to the trunk is no longer essential for diagnosis and treatment, when the patient's tenderness has subsided, when steep postural drainage or prone turning is no longer required, and when there is some spontaneous respiration

The best cuirass respirators are of the "chest-abdomen" type. These, like the tank respirators, act on the diaphragm as well as on the ribs, and so are not only more comfortable than the exclusively "chest" cuirass, but also, in terms of tidal volume, more efficient for any given negative pressure, the lung bases are better ventilated and less likely to develop atelectasis (Cuirass respirators which act mainly on the chest alone achieve their ventilation mostly by sucking the sternum forward; they move the diaphragm very

little and in some cases the diaphragm has even been seen to move paradoxically).

TYPE B. DEVICES

Pressure belts (the Bragg-Paul is the best known) (see fig. 17). These consist of an inflatable bag connected with an electric motor pump. The bag is wrapped once round the patient and inflated so that it just does not hamper his spontaneous inspiration. The pump now increases the pressure in the bag for a few seconds from 16 to 24 times a minute, so squeezing the chest wall. Inspiration is performed by the patient, assisted by the elastic re-expansion of the chest wall. The tidal volume produced is much less than with the negative pressure type of apparatus and pressure belts are inadequate for patients with severe respiratory paralysis. They are, however, of considerable use later when some spontaneous respiration has returned, particularly in cases whose disability consists — as is often the case — in a rigid chest wall with good sternomastoids. They have the advantage that they do not interfere with the action of the muscles of inspiration and the patient has more control of his depth of respiration than when breathing in a cuirass. Further, they are considerably less bulky than a cuirass; they can be left on when not in use and worn while the patient sits in a chair. An exact airtight fit is not required, adjustment is simple, and the belt is relatively inconspicuous.

TYPE C. DEVICES

Positive pressure apparatus for increasing pressure from within the respiratory tract.

(a) *Resuscitators* for use over short periods — e.g. when the tank is opened for nursing or examination. These may be (a) of the type familiar in operating theatres and elsewhere, for the administration of [illegible]

(b) *Prolonged intermittent positive or negative-positive pressure respirators for insufflation through an intratracheal tube.*

There are two designs:

(i) The closed circuit design in which the inspired gases flow

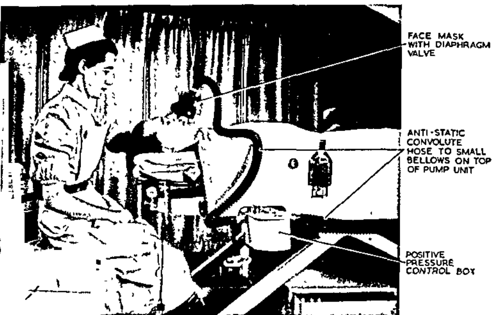


Fig 15 Patient about to receive positive pressure by mask while respirator is open for nursing.

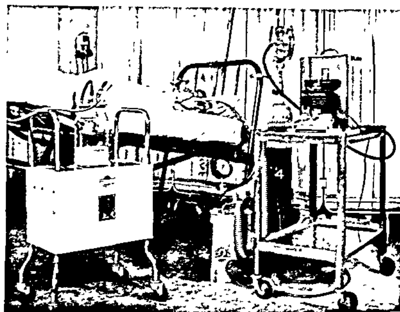


Fig. 18. Intermittent positive pressure respiration being given by intratracheal tube in tracheostome.

Note 1. East pump
2. Thermostatically controlled humidifier (which must be below level of patient)
3 Manometer
4 Oxygen cylinder for emergency
5. Electric suction apparatus. Bed is slightly tilted to assist removal of secretions and prevent atelectases.

from a cylinder into a reservoir bag attached to a sodalime canister for absorption of carbon dioxide; the intermittent pressure is applied to the bag, either manually or by some form of motor. In this type the danger of progressive accumulation of nitrogen in the system and the need to change the soda-lime canister frequently must not be forgotten

(ii) The open type (see fig. 19) in which room air is pumped to a patient by bellows and the expired air passes out through an expiratory valve, or in which compressed gas is fed to the patient and operates a valve changing over the circuit whenever the required inspiratory or expiratory pressures are reached.

These devices require the inclusion in the circuit of a *humidifier* [80, 81] to replace the warming and humidification of inspired air which normally takes place in the nose, pharynx, and upper trachea. It is not sufficient simply to pass gases over or through water at room temperature; for the gases to be humidified the water must be warmed to a temperature at which adequate vaporisation takes place: the humidifier must therefore be fitted with a thermostatic device and the tube between humidifier and patient lagged to reduce condensation Some condensation will, however, take place in the tubing and it is important that the humidifier stands well *below* the patient's level so that this condensation runs back into the humidifier, and not on into the patient's lungs. The tubing leading to the manometer must have its own trap for water condensation. This similarly must be below the patient's head.

A *gasometer* with which to obtain regular readings of the minute volume is also necessary when positive pressure respiration is used for long periods.

Type D. Devices

The rocking bed. This is a motorised version of Eve's rocking stretcher method of artificial respiration in which the diaphragm is moved by changing the direction of pull of the weight of the abdominal contents It consists of a bed which can be rocked through an arc ranging from 5° to about 30-40°, while the patient sist in the Fowler position It should preferably be capable of being started with a rock of less than 5 degrees, which can be gradually increased to the angle required, so as not to frighten the novice. The ventilation obtained is not very high, but has been found to be useful in the convalescent stage for helping the patient to syn-

chronise diaphragm and intercostal or sternomastoid action. Some patients prefer it to the cuirass respirator and can stay on it continuously.

Type E. Devices

Electronic stimulators for artificial ventilation should be mentioned for the sake of completeness. Their application is, however, very limited. Direct stimulation of the paralysed respiratory muscles by faradism is unsatisfactory because in the early stages the stimulus of faradism is intolerable and in the later stages the denervated muscles no longer respond to faradism. For the same reason indirect stimulation of the diaphragm via the phrenic nerve, though it can produce adequate ventilation in the acute stage, ceases to be effective when the nerve fibres degenerate. There are also technical difficulties in its use; particularly it is not easy to keep the electrode in position. There is one theoretical use for this type of stimulation, namely, to suppress the disordered rhythm initiated by a defective respiratory centre.

CHAPTER 9

THE DETECTION AND TREATMENT OF RESPIRATORY COMPLICATIONS

As already mentioned, an *early* recognition that respiratory deficiency, whether it be paralysis of the respiratory muscles or obstruction of the airway, is present, is of prime importance in the treatment of the respiratory complications of poliomyelitis. Haldane's dictum "Anoxia not only stops the machine but wrecks the machinery" should be foremost in the clinician's mind and the sequence of the lungs gradually becoming more and more choked with fluid, and so preventing the gaseous exchange which would stop it, must not be allowed to develop. By the time dyspnoea, cyanosis, and the use of the accessory muscles of respiration is obvious, fluid has usually already begun to accumulate in the chest, making treatment so much more difficult. It is as important to detect that respiratory deficiency exists as to discover the cause of the deficiency.

STAGE 1. DETECTION OF RESPIRATORY DEFICIENCY

Early *general* signs are an unsleeping apprehension, a clammy skin and dilated pupils. A rise in the respiration rate or in the pulse and blood pressure are strongly suggestive, and rates and pressure should be checked hourly if there is the slightest suspicion of respiratory deficiency Reluctance to talk, staccato speech, irregular [illegible]

[illegible]

retention without gross anoxaemia and may be mistaken for a sign of good oxygenation. Gross anoxaemia is, of course, associated with cyanosis.

STAGE 2 DETECTION OF CAUSE OF RESPIRATORY DEFICIENCY

The signs mentioned so far are simply signs of respiratory deficiency, they do not indicate its cause The patient's failure to take adequate breaths may be caused by paralysis of his respiratory muscles, by the danger of aspiration of the unswallowed saliva pooling in his pharynx, or by obstruction of his airway

Is there paralysis of the respiratory muscles?

The signs of respiratory paralysis include

(a) Irregular or asymmetrical movement of the chest, without gross underlying physical signs.

(b) Use of the accessory respiratory muscles, particularly the sternomastoids.

(c) Absence of the pressure normally felt under the costal edge as the diaphragm descends.

(d) Paradoxical respiration: i.e. the upper chest is drawn inwards on inspiration when the intercostals are paralysed and the diaphragm is strong; similarly the lower chest is drawn inwards and the sterno-costal angle narrowed on inspiration if the diaphragm and lower intercostals are paralysed and the upper intercostals or sternomastoids are strong.

(e) A strong downward tug is felt at the larynx during inspiration showing that upward movement of the sternum is not occurring and that the diaphragm is pulling the mediastinum downwards unopposed.

Breathing tests are useful. The simplest test is to ask the patient to count as far as he can in one breath: repetition of this test every hour gives a useful early indication of falling vital capacity. Inability to count beyond 12 indicates severe impairment and requires urgent treatment. Simple vital capacity estimations with a gasometer or spirometer give more exact readings. The normal vital capacity varies of course between individuals; in most men it is over 4 litres, in most women over 3 litres. Readings below 2 litres cause anxiety, particularly if they are falling steadily. At 1000 ccs help is usually overdue. The findings must, of course, be assessed together with other clinical data, but it is much wiser to believe a low vital capacity reading than to attribute it to poor co-operation. The vital capacity can usefully be taken and charted as a routine with the four hourly temperature.

Is swallowing impaired?

Complete paralysis of swallowing is often only a late development. Even a patient with a mouthful of thick saliva, who has been awake all night protecting his airway from aspiration, can sometimes manage to swallow small quantities of fluid, such as a test half-teaspoonful of sterile water, without choking, although, in fact,

secretions have been slowly trickling down the larynx for some time. [illegible]
drink, [illegible]
"vomi [illegible]
the shallow short breaths of a patient who dare not take a deep breath for fear of aspiration from his pharynx.

The defect is detected by means of a stethoscope placed over the larynx. The patient is asked to swallow half a teaspoonful of sterile distilled water and the efficiency of deglutition is checked by the sound produced: patients with normal pharyngeal muscles can swallow this amount with one movement (one sound): patients with a swallowing defect, though they may effectively swallow the water, may require, say, three movements, (three sounds) or they [illegible]

It should be noted that a case with an early or mild swallowing defect may show no signs of interference with his ventilation; yet, unless postural drainage is instituted, he may in fact be in a precarious position, since only a slight increase in paralysis, exhaustion, or the administration of a hypnotic may be sufficient to reduce his voluntary control and lead to sudden aspiration of fluid.

Is the airway obstructed?

Obstruction of the airway is obvious only when there is adequate power in the respiratory muscles; breathing is laboured and loud rhonchi or moist sounds can be heard — often they are more readily localised with the flat of the hand than with the stethoscope. If the power of the respiratory muscles is diminished, obstruction will not be obvious and only careful clinical examination of the chest will elicit and localise the signs. Diminished respiratory excursion and diminished air entry by themselves may well be purely paralytic in origin; but associated with impaired percussion note, mediastinal shift or raised diaphragm dullness, they suggest atelectasis.

A radiograph (obtained with a portable machine) is valuable at this stage, both for diagnosis of the present condition and as a base line for the future, and every effort should be made to have an early X-ray picture as a control.

In assessing the various factors involved in any one case, their inter-relationship must not be forgotten. Until the airway has been cleared, it may be difficult to distinguish deficiency due to respiratory paralysis from deficiency due to "bulbar" paralysis. Only when the airway has been cleared and treatment by postural drainage or artificial ventilation instituted is it possible to assess the relative severities of the two paralyses; either or both may be found to be considerably less severe than at first appeared. Additional points to bear in mind are that (a) coma may cause a patient with only moderate paralysis to breathe more ineffectively than his muscle paralysis alone warrants, and (b) just as a wet sponge is less resilient than a dry one, sodden or atelectatic lung is much more difficult to move than a healthy lung. When the coma is lightened or the condition of the lungs improved, the power of the respiratory muscles may be found to be much more nearly adequate; and when vomiting and excessive secretion are under control the weakness of the pharyngeal muscles may be found to be less dangerous than it appeared to be.

Stage 3. The treatment of respiratory deficiency

Briefly, "bulbar" cases with pharyngeal and laryngeal paralysis are treated by postural drainage and suction (fig. 12), "spinal" cases with respiratory paralysis by artificial respiration in a cabinet respirator (fig. 12 and 13), and both "bulbo-spinal" cases and respiratory cases with superimposed pulmonary complications by both methods combined, i.e. by a sharply tilted cabinet respirator and suction (fig. 13). The indications for tracheotomy with or without positive pressure respiraton are discussed below (p. 84).

Treatment of a "bulbar" case with or without pulmonary pathology. Postural drainage and suction

Posture. The patient is placed in the *semi-prone or prone head-down* position *with his head turned to one side.* The change in posture from *the supine position should be preceded* by very thorough aspiration of any material which may be lying pooled in the back of the pharynx (fig. 10). In this position, saliva or the more dangerous acid vomit, cannot be aspirated far into the respiratory tract, and if aspirated a short way will run out again; at the same time pulmonary secretions, resulting either from the hypercapnic pulmonary congestion or from previous aspiration of foreign material, will also run out into the mouth.

Suction must be applied effectively (see p. 63) repeatedly and conscientiously to remove fluid from the pharynx and cheek, where it will still tend to pool

The patient is kept under constant observation and restlessness [illegible]

cretions, and the morphine group and pethidine because of the supression of respiration and coughing and because of the risk of vomiting.

Fluid Replacement and Feeding. An important problem at this stage is the loss of fluid. Nothing can be given by mouth and the loss by drainage or suction of 1-2 litres of fluid every 24 hours will cause severe disturbance of fluid and salt balance. An accurate fluid chart of both intake and output must be kept from the start so that fluid administration can be modified to the patient's needs. Feeding by stomach tube is not recommended in the early stages, since too often the tube precipitates vomiting, but rectal feeding is favoured by the prone head-down posture and a rectal drip can often be used for 3-4 days. Two bottles of 5 % glucose to one bottle half strength saline is a suitable replacement regime, the rate being modified in accordance with the fluid chart. Initial re-hydration should not be too rapid, since it can cause excessive outpouring of secretions, often sticky at first, which it is difficult to deal with by sucker alone, generally speaking the bias must be towards under- rather than overhydration of the patient, and oedema of the lungs must be avoided at all costs.

In the majority of patients some recovery of the pharyngeal muscles will have begun to occur by the end of 3-4 days. If the danger of aspiration is thought to be sufficiently reduced, feeding by stomach tube can be cautiously introduced, provided all precautions are taken and the patient's reaction is closely observed. The stomach is aspirated before each feed, the residuum is measured and the volume of the next feed correspondingly reduced A high protein diet is gradually introduced. It is remarkable in many cases how suddenly such patients recover. Postural treatment is not completely abandoned, however, until it is felt to be absolutely safe to do so At first the patient is sat up only f [illegible] periods, never alone, and only with every facility [illegible] tipping, should a choking fit start. It is only [illegible] to tide

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Suction must be applied effectively (see p. 63) repeatedly and conscientiously to remove fluid from the pharynx and cheek, where it will still tend to pool.

The patient is kept under constant observation and restlessness is controlled by medication to avoid his reversing his "therapeutic" position. Phenobarbitone is one of the best sedatives at this stage, since it does not depress the cough reflex or the respiratory centre. Bromides are to be avoided because of their stimulation of secretions, and the morphine group and pethidine because of the supression of respiration and coughing and because of the risk of vomiting

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patient over the most difficult stage of his disease and then lose him later through overhasty abandonment of precautions. When feeding is begun it must be remembered that the optimum position is not the prone position where the laryngeal opening lies below the oesophageal, but sitting at about 70° so that gravity helps the bolus to pass posteriorly and downwards.

Patients in whom the rectal drip has to be abandoned before oral or tube feeding can be instituted must be "fed" intravenously. The same fluids are used as recommended for rectal administration, and, in addition, plasma should be given to compensate for the considerable fall in serum albumen which occurs in severe cases. A "gastric replacement" diet with full biochemical control is needed if the condition persists

Intubation of the trachea. When aspiration of saliva or vomit has already occurred more active initial treatment may be required to deal with the resulting pulmonary pathology. Passage of an oral endotracheal tube may be necessary to allow effective suction to be applied lower down in the respiratory tract; it can be very useful in removing obstruction not dislodged by simple postural drainage; it may need to be repeated. Sometimes one such bronchial toilet will succesfully and finally improve the patient's oxygenation. If it is necessary to transport the patient at this stage a tube with an inflatable cuff to block the larynx and prevent any material passing down the trachea can be used, since it is during transport that aspiration is particularly likely to occur. It is not usually recommended to leave such a tube in situ for more than 24 hours for fear of ulceration though longer periods may occasionally be permitted, provided, of course, that the cuff is deflated at regular intervals. It is important to realise that so long as the tube remains in situ, the normal exodus for secretions from the lung is impeded by its presence (it lacks cilia, is of narrower bore than the trachea, and is more easily blocked), and suction is an essential supplement to its use.

Bronchial Toilet under Direct Vision. Finally, in hospital, aspiration under direct vision with a bronchoscope may be necessary for an initial clearing of major obstructions, e.g. plugging of a main bronchus with resulting atelectasis of one or more lobes. It should, however, be emphasised that bronchoscopy in acute poliomyelitis must, unless the patient is already unconscious with anoxemia, be carried out under a short but efficient general anaesthesia, e.g. intravenous barbiturate. The mucous membrane in these cases is

grossly oedematous and local anaesthesia is quite ineffective, with the result that the partly paralysed patient's struggles may make effective bronchial toilet quite impossible, while the completely paralysed patient, who is unable to resist, suffers such pain and terror that adults have been known to exact from those in charge a promise that the performance shall never be repeated. Readers should perhaps again be reminded of the curare-like effect of acute poliomyelitis in which the patient can feel everything but resist nothing, and sometimes cannot even speak.

TREATMENT OF A PATIENT WITH PARALYSIS OF THE RESPIRATORY MUSCLES.

As already mentioned, assisted respiration should be instituted before respiratory defect is obvious and anoxemia and hypercapnia have brought about their own pulmonary changes.

Control of Artificial Respiration

The aim of artificial respiration in poliomyelitis is to give the patient — often over long periods — a ventilation similar to that which he would give himself in order to maintain full oxygenation, adequate carbon dioxide elimination and a normal blood pH.

In the presence of complete or severe respiratory paralysis this aim can only be achieved either by negative pressure respiration with a cabinet respirator *or* by positive pressure (e g. by a hand bellows or by resuscitation apparatus) to mouth or intratracheal tube. Since positive pressure by mouth or oral intubation cannot be maintained indefinitely and a tracheostome is to be avoided if possible (see p. 82), a negative pressure cabinet respirator, and understanding of its use, are essentials in the treatment of respiratory poliomyelitis in the acute stage when respiratory paralysis is, or may become, severe.

In a cabinet respirator adequate ventilation is usually maintained for a normal adult — at rest, afebrile, with a normal metabolic rate, normal lungs and no obstruction to his airway — by a pressure change of — 15 cm H_2O at a rate of 15 respirations per minute. Approximately the same pressure change, is suitable for patients of all sizes (excluding the newborn) because the actual force exerted depends on the pressure *and* the area over which it is applied Pressure changes of less than 10 cm. have little effect, while the need for higher changes is strongly suggestive either of obstruction to the airway (pathological, or legitimate, as when

there is an endotracheal tube *) or of pulmonary pathology (atelectasis, pneumonia or oedema).

A pressure change of 15 cms. H_2O will give a tidal air of about 500-600 ccs. in a man, and 400-500 ccs. in a woman, and the corresponding minute volumes (tidal air x rate of respirations per minute) will be, at 15 respirations per minute, 6-8 and 5-7 litres respectively. A child of five years receiving the same pressure at the more natural rate for his age of 20 respirations per minute will have a tidal air of about 200 ccs., or 4 litres per minute. If there is severe respiratory and locomotor paralysis with marked wasting, the requirements may later fall far below these figures, and even adults may eventually be adequately ventilated for considerable periods with, for example, tidal airs of only 200-300 ccs., a fact which obviously plays some part in the subsequent weaning of cases to less cumbersome respiratory apparatus and to independent respiration.

The main pressure change given in a cabinet respirator is a negative one. Many of the older type respirators can, in fact, give only a negative pressure change, but more recently positive pressure control valves have also been fitted, and a smaller positive component can be introduced if required, e.g. to counteract the tendency of the chest to be drawn into increasing expansion and to assist the drift of the secretions towards the pharynx. Any positive pressure must, of course, be compensated by a corresponding reduction of the negative pressure to give the same total pressure change, e.g. 0 to —15 or +5 to —10. Positive pressure is, however, not always well tolerated; it tends to be resented, particularly after a meal, when the bladder is full, and when the patient is head-down.

While the required *pressure change* tends to be fairly constant, the *rate* should be adjusted with some regard to the expected rate of spontaneous respiration. (The patient's rate before transfer to the respirator is of course no guide). Spontaneous respiration is faster in young children than in adults, in fever (since the respiratory exchange is increased) and in pulmonary complications (which reduce the area of lung parenchyma available for respiratory exchange). In these conditions, therefore, the rate of the respirator will often need to be increased, (to be slowed again as the complications subside). It should however, be remembered that

* An endotracheal tube, particularly if used with a right-angled connection can reduce the effective pressure by 5 cms H_2O — or more — and the pressure in the cabinet will have to be adjusted accordingly

doubling the rate will not, double the respiratory exchange; each breath contains a proportion of dead space air and an increase in the number of respirations per minute increases the amount of dead space air in a given minute volume; in addition, at the faster rates the pressures act for a shorter period, with the result that the tidal [illegible] against the use of heroic pressures or rates since these are unphysiological and may rupture alveolar walls.

Tests for adequate ventilation

The mangement of artificial respiration would be greatly simplified were there rapid and fool-proof tests for the oxygenation of the patient and for the carbon dioxide reserve. Unfortunately such tests are not readily available and in practice clinical judgment remains our main guide, with biochemical estimations as a subsidiary aid.

Underventilation causes hypoxia, carbon dioxide retention, respiratory acidosis, a rise in systemic and pulmonary arterial pressures, pulmonary transudation and atelectases. Clinically it appears as a rising pulse rate, a raised blood pressure, anxiety and sweating; the patient feels he is "not getting enough" secretions increase and the respiratory excursion decreases in relation to the underlying pulmonary pathology, which may be either cause or effect of the under-ventilation. Supplementary oxygen gives temporary relief but it does not correct the carbon dioxide retention. And the need for supplementary oxygen is itself an indication of underventilation.

Over-ventilation leads to excess loss of carbon dioxide and respiratory alkalosis, and tetany may occur. Clinically it is not always easy to diagnose and it may even be difficult to distinguish moderate degrees of overventilation from underventilation, particularly as the overventilated patient may still complain that he is "not getting enough". Useful indications are· an adequate tidal air, absence of pulmonary pathology and a low serum bicarbonate.

The most useful subsidiary tests for ventilation are

1 Estimation of the carbon dioxide combining power of the blood. This is the most generally useful test, since it can be done on the venous blood and can be repeated after a few hours to show the effect of any changes.

2. Estimation of the pH of the blood (arterial or capillary) requires special procedure for collection of the specimen and is of limited clinical application. Repeated arterial puncture is usually undesirable.

3. Photoelectric estimation of the oxygen content of the blood in the ear by an oximeter. In experienced hands this method is useful but for the uninitiated it can be misleading.

4. Estimation of the pH of the urine — this is only useful as a very rough guide to over- or underventilation over long periods; the urine will of course probably have been secreted over 3-4 hours and during that time considerable variation may have taken place in the biochemistry of the blood. The decomposition of urea to ammonia must also be remembered.

[illegible]

the source of the trouble so that prompt action can be taken to clear the chest by physiotherapy and posture, and if necessary bronchoscopy, using positive pressure respiration by face mask and if necessary supplementary oxygen to tide over the crisis.

The majority of patients dependent upon respiratory aid over long periods will be found to be technically overventilated as judged by their serum bicarbonate which often varies around 17-18 meq. (44-45 vols. %). Attempts to raise this level by decreasing the pressure or rate usually leaves the patient subject to bouts of deficient ventilation, related presumably to temporary increased oxygen requirement which cannot be foreseen. As the patient's spontaneous respiration gradually returns the serum bicarbonate level gradually rises, but it may remain for many months at the lower end of the normal scale.

Transfer to a Respirator. (Fig. 19) Before the patient is transferred to the respirator every preparation is made in advance to ensure that his transfer shall be as quick and efficient as possible The nursing team should already have been trained on a dummy

The motor of the machine is started and the rate is adjusted in advance to the expected requirements of the patient The valves should be fully opened. The lamps are turned on to ensure the cabinet and mattress being warm, and the foot board and other adaptable accessories are adjusted to the size of the patient. A neck seal large enough to go over the piece of lint which is wrapped smoothly round the patient's neck in advance (gamgee tissue usually breaks up into lumps) is chosen. Finally, sufficient help is collected for moving a completely helpless patient to whom every disturbance is acutely painful, at least four people are required to move a paralysed adult If the cabinet has any form of split front the patient is transferred straightaway

to the correct position with his neck resting on the lower half of the front If the front is not split he must be moved in two stages — first he is transferred to the cabinet's stretcher and then the nurse looking after his head moves round to pass her hands through the hole to take the head again and deliver it through the hole, as the patient is lifted up the stretcher

Every care is taken to adjust the neck seal to exclude draughts and avoid friction round the neck, since this is the place where pressure leaks are most likely to occur and the patient is likely to suffer most discomfort At the same time others adjust the limbs to the optimum posture for the avoidance of deformity and pressure sores, and blankets are tucked in securely The older types of machine have no baffles over the control valves and the bedclothes should be adjusted with this in mind

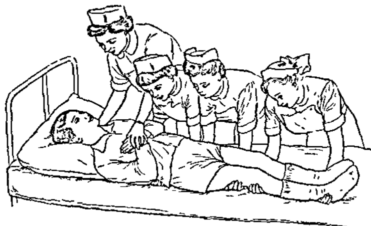


Fig 19 Patient ready to be transferred to respirator Four people are needed to lift an adult patient in the acute stage The senior looks after the head and directs operations

During transfer to the lung, and subsequently for nursing, it may be necessary to give the patient assisted respiration by mask from a resuscitator. In some machines this can be done from a special attachment worked by the same motor — if not it is advisable to leave the motor running so that the same rate is used (There is a natural tendency to increase the rate of artificial respiration given manually)

Once the patient is in the respirator and the rate and pressure of ventilation have been satisfactorily adjusted to the patient's needs, the problem is largely one of nursing, and the same as has been described in Chap. 7, except that particular attention must be paid to preventing hypostasis and accumulation of secretions

in the lungs. The arm ports which seal round the nurse's arm are useful for minor nursing adjustments, for taking the pulse, etc;

in the cabinet is near zero; the nurse should therefore turn the face of the pressure gauge to face her, whichever side of the cabinet she is working. For procedures which cannot be carried out with the respirator closed, positive pressure by face mask or mouthpiece, with or without supplementary oxygen, is used if the patient cannot tolerate even short periods without assistance; or the patient may prefer manual compression of the chest. (Fig. 15). The temptation to "scamp" treatment of skin, mouth, and limbs, must be resisted since it can only lead to greater difficulties later, but the best use should be made of each disturbance of the patient so that he has intervals for sleep and rest. This is particularly important in children, who are usually acutely apprehensive of disturbance and also suffer severely from loss of sleep. Adults, on the other hand, often hate to be left alone and benefit from constant minor adjustments. Throughout, particular attention must be paid to preventing hypostasis of the lungs. Regular changes of posture, e g. alternating semi-lateral position and changes of the tilt of the respirator, coupled with intelligent use of a stethoscope to detect early small areas of *atelectasis and so direct the appropriate postural drainage*, will usually suffice to maintain a healthy condition of the lungs

For the first two or three days in a respirator patients often find

position is not easy at the best of times, and the remorseless rhythm of the respirator often makes patients choke. (Choking in these ... evidence of paralysis of swallowing.)

n arm port

to drink.

Usually after this time the technique of swallowing has been acquired but the greatest care on this matter is essential to prevent aspiration of food, drink, and vomit. If a "choke" is suspected at any time the cabinet should be tilted straightaway into the head-down position and an armport opened momentarily, since the patient cannot clear his own airway by coughing, or resist aspiration further into the respiratory tract by the respirator.

Early signs of swallowing defect must of course be watched for throughout the period of spread of the paralysis, since such a defect will convert the simple respiratory (or spinal) case into a "bulbo-spinal" case.

3. Treatment of a patient with combined respiratory and pharyngeal paralysis — a "bulbo-spinal" case.

These are usually patients with considerable locomotor paralysis as well. They are treated by a combination of the two methods described above, namely assisted ventilation and postural drainage. Constant watch must be kept on the condition of the lungs. The prone or semi-prone head-down position is the most efficient posture for drainage.

This position may however be impossible in a cabinet respirator because of soreness of ribs, sternum or shoulders, venous congestion or simply too short a neck, or because of the design of the respirator, and it is usually necessary to use the steep head-down *supine position sometimes with the whole respirator tilted* on blocks to increase the slope The weight of the patient on his neck and shoulders presents a considerable nursing problem which can be partly solved by taking some of the weight off the shoulders by a band round the pelvis.

These cases present a definite nursing challenge, which if successfully met will allow the patient to be tided over the relatively short period of severe swallowing defect and enter the chronic stage without the serious handicap of a tracheotomy or the development of changes in the lungs. Antibiotics should be given, preferably prophylactically, or at the very first suggestion of pulmonary infection Small areas of atelectasis are treated by postural drainage, compression and percussion of the chest and assisted coughing Bronchial dilators of the nor-adrenaline group (e.g. isoprenaline) or antihistamines (e g. phenergan) which have also a bronchodilator action can be useful, but care should be taken that postural drainage is correct and suction effective before they are administered, since they may have a surprisingly rapid clearing effect. A four-hourly administration of trypsin by aerosol can also be useful given half an hour before physiotherapy. Larger areas of atelectasis may necessitate clearance by bronchoscopy, even repeated two to three times.

Occasionally, however, cases occur in which secretions are so

excessive that they cannot be controlled by posture and peroral suction. In these tracheotomy will have to be performed as a life-saving measure to enable adequate bronchial toilet to be effected and to shut the airway to vomit and saliva coming down from above.

Tracheotomy

The *advantages* of a tracheotomy are:

1. Repeated toilet of the bronchial tree is possible by suction through the tracheostome.

2. The cuff of the tube prevents aspiration of saliva and vomit in cases with paralysis of swallowing: it therefore obviates the need for steep postural drainage in a tank respirator.

The *disadvantages* are:

1. The patient is deprived of his voice: this can be a cruel imposition on a patient already rendered helpless by the disease and entirely dependent on talking for active contact with his environment.

2. The exacting demands of the patient for skilled medical and nursing attention are, if anything, increased rather than decreased by the presence of a tracheostome. It will be noted that the normal drift of the patient's secretions towards the throat is interrupted by the presence of the tube and the entire responsibility for their removal now rests on the patient's attendants. Consequently the problem of secretions and danger of the development of pulmonary infection and particularly of atelectases is prolonged indefinitely — in practice until some time after the tube is removed.

3 Contrary to expectation, respiratory crises continue to occur and after tracheotomy is performed repeated bronchoscopy may still be necessary, particularly if those in charge have not already considerable experience of the method. Some pulmonary pathology is almost inevitable with a tracheostome.

4 The by-passing of the mucous membrane of the upper respiratory tract allows the air to reach the trachea dangerously dry and cold, and some method of humidification is necessary.

5. Persistence of severe respiratory paralysis can make closure of the tracheostome difficult or even impossible and so prolong — sometimes for life — the patient's period of dependence upon skilled attention.

6. If intermittent positive pressure respiration is used, the positive pressure in the chest can significantly hamper the venous return and so increases the risk of "shock" and vasomotor disturbances in the acute stage.

7. The need for biochemical control seems to be greater with this type of respiration

The decision to make a tracheostome must therefore not be taken lightly in acute poliomyelitis, for fear that the immediate advantage of easier bronchial toilet and readier access to the patient will later be heavily outweighed by the development of pulmonary pathology, and the distressing inconvenience of a permanent tracheostome with its perpetuation of the problem of pulmonary secretions and of the patient's need to remain in hospital. It should be obvious that there is no place for "prophylactic" tracheotomy in acute poliomyelitis In a few cases of combined bulbar and respiratory paralysis, however, tracheotomy will be necessary because of inability, for one reason or another, to keep the airway clear

1 *The tracheostome* This is made under general anaesthesia, with an oral endotracheal tube in situ, directly below the thyroid cartilage

2. *The cuffed tube* This should be wide enough to allow free respiration (size 9 is recommended for an adult) and as short as possible, i.e. cut off a few millimetres below the cuff to avoid its extending beyond the carina, if it is too long it will find its way into a (usually the right) bronchus with resulting hypoventilation and atelectasis of the remaining lung The tube is secured by tape round the patient's neck When secretions are minimal the tube should be changed at least once a week when they are increased it may be necessary to change it more frequently, otherwise secretions will accumulate round the tube causing either local inflammation, crusting or, when they run back into the bronchi as the patient sits up, atelectases The tube should be lubricated very thinly before insertion

The cuff of the tube is inflated with an ordinary syringe using the guide balloon as the indicator of its tension, only the least pressure compatible with prevention of the entry of material from the pharynx and the loss of air towards the pharynx is used, the patient himself is the best guide to the tension Sloughing of the trachea is a real danger and the cuff must be deflated at regular intervals, e.g. every 4 hours, during which time the patient can, if this is still indicated by the bulbar paralysis, be tipped head-down while artificial respiration is applied by hand to the chest wall

3 *The endotracheal connection* (for connection with an intermittent positive pressure pump) Either a simple connection of Nosworthy type or a curved tube with a straight side tube for suction is suitable When the positive pressure apparatus is disconnected for spontaneous respiration the open end of the endotracheal tube must be lightly covered to prevent inhalation of dust and other foreign material into the trachea, and to retain a pool of warm moist air round the opening

Intermittent Positive Pressure Respiration In the presence of a

tracheostome artificial respiration can be given in a cabinet respirator with the neck seal pressed inwards by a flat piece of metal, or by an intermittent positive or negative-positive pressure pump.

The principles of positive pressure respiration itself differ little from those of negative pressure respiration, i.e. the rate and range of pressures aimed at are similar, though the pressure change is now mainly positive and slightly less, e.g. + 12 or 13 mm. H_2O. The ventilation can be estimated by temporary inclusion of a spirometer in the circuit. The apparatus should allow the tidal air to be varied as well as the rate and pressure. A sudden drop in the minute volume (in other words, the need for an increase in pressure to maintain it) suggests the development of secretions or atelectasis (if defect of the apparatus is excluded) When these have been overcome the ventilation should be reassessed. Too rapid a loss of CO_2 by hyperventilation can convert hypertension to hypotension and a dangerous state of shock may ensue very rapidly.

The need for removal of secretions by suction, postural drainage and constant shifting of the patient is, with intermittent positive pressure respiration almost continous. Routine changes of posture combined with physiotherapy to the chest wall (squeezing the chest, assisted coughing, vibration and tipping) to dislodge [illegible] and

[illegible]

minimum which will maintain a healthy state of the lungs. But always a close watch must be kept on the chest, and small areas of atelectasis (often detected earlier with the stethoscope than with the radiograph) cleared as soon as they develop.

For weaning from respiratory aid see Chapter 14.

CHAPTER 10

PREGNANCY IN POLIOMYELITIS

The particular susceptibility of the pregnant and lactating woman to the poliomyelitis virus means that pregnancy and lactation are rather frequent complications ([51, 810]).

In the acute stage the risk to the foetus appears to be similar to that in other acute fevers: abortion and premature labour may occur and there is danger of foetal abnormality if infection occurs before the placental barrier is established. If the foetus survives the acute stage and there is no involvement of the respiratory muscles the pregnancy can usually be allowed to take its normal course. If there is respiratory involvement the decision to allow the pregnancy to continue is influenced by the severity of the respiratory paralysis and by the stage of the pregnancy. This severe [illegible] decision. A milder degree of respiratory paralysis later in pregnancy indicates a considerable chance of obtaining a normal live baby and the increasing ventilation requirement and mechanical interference with the movement of the diaphragm will usually be matched, though not necessarily outpaced, by the improvement in the respiratory muscles. If, however, the patient begins to show severe respiratory distress labour should be induced before her condition deteriorates further

The uterine muscle is not usually affected, provided oxygenation is adequate, and vaginal delivery is usually possible in both the acute and later stages, if there are no other contra-indications. Prolonged labour is of course undesirable, and a small baby is an advantage. There is usually marked improvement in respiratory function after delivery.

Protection of the pregnant and lactating woman against infection with the poliomyelitis virus, and protection of the baby both deserve careful consideration. A pregnant and lactating woman who is a known contact should be given passive immunisation with gamma globulin, while pregnant women in general should be regarded as a priority group for active immunisation wherever vaccine is available.

Similarly, the need for protection of the baby must be considered.

If it is delivered too early to acquire antibody from its mother it requires protection against virus transmitted from the mother — in utero or during delivery — and from the mother's contacts, who may be expected to be infected. Cases of both intra-uterine and neonatal infection have been reported and passive protection with gamma globulin should be given where the time relation suggests risk to the baby.

There have been several tragic outbreaks of acute poliomyelitis in maternity homes, and obstetricians and midwives should be aware of the importance of taking precautions when in contact with poliomyelitis cases — and their contacts — in order to avoid transmitting the virus to the own particularly susceptible clientele.

CHAPTER 11

THE TRANSITIONAL STAGE

The acute stage is considered to last from two to three weeks During it we have had to deal with a more or less ill patient suffering from a febrile virus infection which attacks particularly the central nervous system and is associated not only with locomotor paralysis but also with paralysis of certain important functions, swallowing, respiration, micturition, and bowel function, with the resulting [illegible]

restored to normal health. There is still inflammation of the central nervous system — manifested *clinically* by persisting meningism, lethargy and/or irritability, and "tiredness" and *pathologically* by persistence of generalised inflammatory changes in the central [illegible]

develop and change will all be in the direction of recovery

This transitional stage varies from one to eight weeks following the three weeks of the acute stage Its actual duration in any one case appears to depend less on the severity of the paralysis than on the severity of the encephalitis and meningism [illegible]

[illegible] small group of muscles, will usually have a short transitional period and may recover his feeling of well-being within three to four weeks of the onset, regardless of the recovery or persistence of the paralysis In contrast, the patient who has had a severe meningism and evidence of widespread distribution of the virus shows a more prolonged transitional period, whether or not there is much residual paralysis; the meningism subsides only gradually and the patient by no means feels fit for his normal activities — still less for struggling with newly acquired disabilities — for eight to twelve weeks. Cases with reduced ventilation are of course still longer in recovering their sense of well-being.

It is important to recognise the existence of this transitional period because of its bearing on management. There has been a tendency in recent years to try to hurry the patient over this period; to start vigorous exercises early, to urge him to sit and stand up, and even to send him home at a stage when all his own instincts are to rest. It is therefore necessary to stress that the disease is one of the whole central nervous system and not simply a lower motor neuron paralysis. Even the patient who has suffered only the non-paralytic form of the disease requires, like other patients recovering from other forms of encephalitis or meningitis, a period of convalescence before returning to full activity. Still more respect should be paid to the need for rest in the patient with associated lower motor neuron involvement, since for him even apparently minor activities can demand quite exhausting effort and concentration.

It is, in fact, not only pointless but on occasions cruel and dangerous, to expect and urge a patient to perform exercises and even (as is too frequently the case) to start walking so long as meningism persists — pointless, because normal posture is impossible in the presence of meningitic spasm, whether there is accompanying paralysis or not; cruel, not only because it is exhausting and painful at this stage, but also because the patient gets the impression that he is more severely disabled than he is; if the same attempt is made a few weeks later the disability of the meningism will have vanished, normal postural reflexes will no longer be inhibited and time will have been allowed for a good deal of true recovery to take place (see p. 90). If one's quadriceps is going to recover within a few weeks there is after all little purpose in trying to learn to walk without it.

The most important reason against strenuous treatment at this stage, however, is that it is dangerous to the affected nerve cells. There is considerable clinical evidence that "fatigue" in the early stages, while the nerve cells are still disordered following invasion by the virus, not only delays their recovery but may also cause returning power to disappear — a phenomenon once seen never forgotten! [82, 83].

The treatment recommended for the transitional stage is therefore twofold: (a) improvement of the patient's general condition, and (b) continued careful management of all his paralysed parts, on the lines described for the acute stage, but now allowing a gradual increase in activity, the main burden of effort still being taken by the

[illegible]

only two or three more concentrated periods of treatment in which the patient's efforts are gradually [illegible]. Reducing the frequency [illegible]

[illegible]

depressing — disabilities.

guide on prognosis can be obtained by considering the severity of the paralysis of other muscles of similar cord innervation: severe paralysis of other muscles of related innervation suggests that much of the damage done by the virus will be permanent. The rate of recovery of muscle power is most rapid during the first six months and falls off gradually over the following six months so that *under favourable conditions* most recovery of muscle *power*, as tested by muscle charting, will near completion by the end of the first year [81]. Conditions are, however, by no means always favourable and examples of exceptions to this rule are numerous. Unfavourable conditions which may postpone recovery include:

(i) widespread severe paralysis which postpones the time when the individual muscle under consideration can work under optimal conditions and receive the attention it requires;

(ii) stiffness, particularly contracture of the antagonists, which increases the load and often the mechanical disadvantage of the muscle;

(iii) respiratory paralysis, which may severely limit the ability of both patient and physiotherapist to concentrate on an individual paralysed muscle.

(b) *Recovery of muscle function.* This recovery is not reflected on the muscle chart. It embraces not only recovery of muscle *power* but also recovery of *endurance* (which is strikingly lacking in the early recovery stage of poliomyelitis) *co-ordination*, *skill*, and *confidence*. It continues for a good deal longer than simple recovery of power; from the point of view of a patient with severe extensive paralysis it may be greater in the second year than in the first, or even in the third year than in the second. Thus he may learn to breathe independently in a chair during the second year, and to manoeuvre from bed to chair and from chair to toilet or to car only in the third year. In a case such as this only by the end of the third year will he have gained independence, and further useful improvement of function can continue even after these major objectives have been achieved.

Children also fail to conform to hard and fast rules about duration of functional recovery. As they grow they may show unexpected improvement which, though probably small in itself, may be sufficient to enable them to abandon apparatus previously essential or to take part in activities which were previously impossible.

The question which is perhaps most commonly asked regarding

the physiotherapy and rehabilitation of poliomyelitis is· for how [illegible]

long as it is helping the patient usefully to improve his locomotor or respiratory function. It should *not* be continued indefinitely after it ceases to do so. Generally speaking, the period over which treatment will be useful is shorter, the more mild and the more localised the paralysis, and longer, the more severe and the more widespread it is. Some patients benefit from occasional periods of intensive [illegible]

itself — when combined with other activities. Children particularly may benefit from intermittent out-patient treatment confined to the holidays, rather than from treatment continued through the school term when it may seriously interfere with their education, of which the importance in the seriously disabled must never be underestimated.

CHAPTER 13

MANAGEMENT OF THE RECOVERY PERIOD

The picture presented by the paralytic patient has now changed from that described at the beginning of Chapters 7 and 10. Unless there is still severe respiratory impairment, or a superimposed urinary or respiratory infection he is rapidly recovering his sense of well-being. Pain and tenderness are reduced to discomfort at the outer ranges of joint movement and mild tenderness in the *muscles, and the case of acute virus infection is now a case of lower* motor neuron paralysis whose disability depends on the severity and distribution of the damage to the motor neurons in the cord, and on any residual deformity which may have developed during the acute stage. During the transitional period he will have been transferred from the isolation of the infectious diseases hospital or ward to the greater freedom of the orthopaedic hospital. Here both the emphasis on rehabilitation and the subsidiary social activities will help to encourage him that he has really begun to move towards home. If he is a child he will have begun to take part in the ward's school activities — at first passively and then, if his disability allows, more actively. If he is an adult he will have benefitted from renewed contact with the outside world and will have taken note of the achievements of those whom he sees to be either as or even more severely disabled than himself. In this atmosphere he is prepared to devote all his energies to developing to the maximum the power which remains to him.

The management of the recovery period begins with *careful examination and recording of the condition of the locomotor system.* The record so obtained provides both a base line against which recovery (or deterioration) can be assessed and a ready reference for the planning and subsequent modification of treatment. Brief reference to the record will provide the detailed information which could only otherwise be obtained by repeated and tiring re-examination of the patient.

This examination of the locomotor system falls into two main parts: (a) testing of the muscle power and (b) testing of the passive range of movement at the joints and detection of muscles which still shew resistance to stretch.

Chart for recording paralysis of cranial nerves and respiratory defects

Examiner									
Date									
		R	L	R	L	R	L	R	L
Diplopia	III, IV, VI								
Nystagmus	III, IV, VI								
Palate	V								
Masseter	V								
Deviation of Jaw	V								
Facial	VII								
Swallowing	IX								
Elevation of Larynx	C 1 2 3								
Voice	X								
Sternomastoids	XI								
Diaphragm	C 3 4 5								
Latissimi dorsi	C 6 7 8								
Upper intercostals	T 1—6								
Lower intercostals	T 7—12								
Abdominals	T 8—12								
Findings on X-ray screening of chest									
(a) Sternomastoid movement									
(b) Separation of upper ribs									
(c) Separation of lower ribs									
(d) Diaphragm									
Cough									
Vital capacity in ccs									

Chart for recording paralysis of neck and upper limb (a less detailed chart is of course sufficient in the acute stage of poliomyelitis)

Muscle	Segments	R	L	R	L	R	L
Examiner							
Date							
Sternomastoid	XI and C 2 3						
Anterior cervical	C 1—6						
Posterior cervical	C 1—6						
Trapezius	XI and C 3 4						
Pectoralis major	C 5—T 1						
Pectoralis minor	C 7, 8 T 1						
Latissimus dorsi	C 6 7, 8						
Serratus anterior	C 5 6, 7						
Deltoid — anterior	C 5, 6						
medial							
posterior							
Coracobrachialis	C 7						
Supraspinatus	C 5 6						
Infraspinatus	C 5 6						
Subscapularis	C 5 6						
Teres major	C 5, 6						
Teres minor	C 5						
Rhomboids	C 5						
Levator scapulae	C 5						
Biceps	C 5 6						
Brachialis	C 5, 6 7						
Brachioradialis	C 5 6						
Triceps	C 6 7 8						
Supinator	C 5 6						
Pronator	C 6 7						
Flexor carpi radialis	C 6 7						
Flexor carpi ulnaris	C 8—T 1						
Palmaris longus	C 8						
Extensor carpi radialis longus	C 6 7						
Extensor carpi radialis brevis	C 6 7						
Extensor carpi ulnaris	C 7 8						
Flexor digitorum sublimis	C 7 8—T 1						
Flexor digitorum profundus	C 8—T 1						
Flexor pollicis longus	C 8—T 1						
Extensor digitorum communis	C 7 8						
Abductor pollicis longus	C 7 8						
Extensor pollicis brevis	C 7 8						
Extensor pollicis longus	C 7 8						
Interossei dorsales 1, 2 3 4	C 8—T 1						
Interossei palmares 1 2 3	„						
Lumbricales 1 2 3 4	„						
Adductor pollicis	„						
Flexor pollicis brevis	„						
Opponens pollicis	„						
Abductor pollicis brevis	„						
Hypothenar muscles	„						

Chart for recording paralysis of trunk and lower limbs

Examiner

Date

Muscle		Segments	R	L	R	L	R	L
Rectus abdominis	Upper	T 6—9						
	Lower	T 10—12						
External oblique		T 7—12						
Internal oblique		T 7—12 L 1						
Quadratus lumborum		T 12 L 1 2 3 4						
Sacrospinalis								
Upper thoracic		T 1—6						
Lower thoracic		T 7—12						
Lumbar		L 1—5						
Hip flexors		L 2 3 4						
Sartorius		L 2 3						
Internal rotators		L 2 3 4 5 S 1						
External rotators		L 4 5 S 1 2						
Adductors		L 2 3 4 5						
Tensor fasciae latae		L 4 5 S 1						
Gluteus medius		L 4 5 S 1						
Gluteus maximus		L 5 S 1 2						
Quadriceps		L 2 3 4						
Semimembranosus		L 4 5 S 1						
Semitendinosus		L 4 5 S 1 2						
Biceps		L 5 S 1 2 3						
Calf		L 5 S 1 2						
Tibialis anticus		L 4 5						
Tibialis posterior		L 4 5						
Peroneus longus		L 5 S 1						
Peroneus brevis		L 5 S 1						
Extensor hallucis longus		L 5 S 1						
Extensor digitorum longus		L 5 S 1						
Extensor digitorum brevis		L 5 S 1						
Flexor hallucis longus		L 5 S 1 2						
Flexor digitorum longus		L 5 S 1 2						
Intrinsics		L 5 S 1 2						

Testing of muscle power. Specimens of suitable muscle charts are shown on pp. 96-97. The muscles are grouped according to the joint on which they work. For a number of reasons, it is necessary to test muscles rather than movements: one muscle may have more than one function; one function may be served by two or more muscles whose imbalance, if not corrected, can be as great a disability — particularly in children — as complete paralysis; weakness in performing a joint movement may be caused not by weakness of the muscles but by increased resistance to the movement, e.g. by spasm or the contracture of the antagonists: finally, the use of tendon transplantation in subsequent reconstruction makes it important to have a record of the development of the strength of the individual muscles during treatment. It is therefore important to examine the strength of the muscles themselves, not only by feeling the strength of the movement they perform, but, wherever possible, also by feeling the hardness of the muscle's contraction or the tightness of its tendon.

In Great Britain and a number of other countries a modification of Lovett's system of manual grading of muscle power is in use. In the modification recommended by the M.R.C. six gradings of muscle power are used, the grades being determined by the muscle's ability to perform its normal function of moving or fixing the part on which it acts. Grade 0 represents no power, Grade 5 full power, and there are four intermediate grades:

0 = no contraction.
1 = a flicker of contraction — insufficient to move the joint.
2 = contraction sufficient to move the joint with gravity eliminated.
3 = contraction sufficient to move the joint against gravity.
4 = contraction sufficient to move the joint against gravity and some resistance.
5 = full power.

This method of grading has the advantage that it is applicable to most muscles regardless of their size or of the age, size or sex of the patient; it requires no special apparatus; and being based on function it requires neither interpretation nor the memorisation [illegible] coarse, but for [illegible]

be explained to the patient who may otherwise be depressed by the

failure of even considerable clinical improvement to be reflected in the muscle chart.

This type of "manual" grading is unsuitable to clinical research. Studies on the effect of different activities or training routines require more sensitive methods of measurement of power and various methods are available. One can record the weight which can be lifted a certain number of times through a certain distance; or one can measure the force which can be exerted on a dynamometer. The sensitivity of a dynamometer used for this purpose should be variable through a relatively wide range and it should record the strength of both static and dynamic contractions. A strain guage dynamometer is particularly useful for the measurement of muscles which are too weak for weight lifting ([85]).

The more accurate methods usually have the disadvantage that they are applicable to a relatively small number of muscles, and there is a risk that their sensitivity may be greater than the errors of positioning, etc. inherent in the method warrant. Within these limits, however, they make it possible to obtain recordings of power comparable from session to session and from patient to patient.

2. *Examination of passive joint movement* The limitation of passive joint movement at this stage of poliomyelitis is caused by resistance to stretch by the muscles which cross it and not by true joint stiffness (which in poliomyelitis is a late secondary development). *The examination should therefore include not* only estimation of the range of movement, but also detection of the muscles responsible for the limitation so that further efforts can be made to relax them and so restore the full range of movement This fact — that the stiffness is produced by muscle resistance — can be readily demonstrated when the muscle at fault is one which crosses two joints; thus a tight calf muscle *which* prevents dorsiflexion of the ankle *when the knee is straight* can be relaxed by flexion of the knee and full dorsiflexion of the ankle easily obtained. This phenomenon can be used to maintain a normal range of joint movement during the period over which the resistance of the muscle is being overcome.

Muscles in which such tendency to shortening should be especially sought because of the severe disability which may result from the contracture which will develop if it is not controlled are the following

(a) The adductors of the arm. Contracture in these muscles renders any residual power of abduction of the arm useless; if the *pectoralis major is one of the muscles affected* the movement of the ribs of that side will be restricted.

(b) The spinal muscles. Severe scoliosis can develop as a direct result of contractures in either the long or the short muscles of the spine; it can also develop secondarily as a result of imbalance and deformity of the lower limbs.

(c) The muscles acting on the hip joint. Any of these can develop contracture if paralysed, e.g. the ilio-psoas, with the resulting flexion, external rotation and abduction deformity of the hip and associated apparent shortening of the limb, which alters the whole mechanics of locomotion; similarly, shortening of the ilio-tibial band is a common source of deformity at the hip, particularly where the quadriceps is also paralysed.

(d) The calf muscles. The equinus deformity of the foot with its associated apparent lengthening of the limb, which results from contracture or overactivity of the calf muscle, is perhaps the most familiar sequela of poliomyelitis. Correct splinting and hot packs in the early stages, and constant supervision and correction *through the period of growth are necessary if this very* obstinate and disabling tendency is to be controlled.

(e) The dorsiflexors of the ankle. Relative overactivity of the dorsiflexors in the presence of a paralysed or very weak calf can produce the reverse deformity of a calcaneus foot. In adults it is wise to limit the range of movement to a few degrees short of the neutral position (with the knee straight). In children, however, one is reluctant to encourage any contracture so long as growth, with its tendency to increase a deformity, persists. A fuller range of elasticity of the calf is therefore desirable in children.

These contractures have been selected for special notice because their development so frequently, and sometimes so grossly, increases the disability of the paralysis. To call attention to them may therefore reduce the number of cases in which the initial tendency of the muscle to resist stretch develops *into a true* contracture — either immediately following the acute stage or, in children, at any time during growth. Both contracture and excessive laxity can of course develop in any muscle which is denervated or partially denervated.

With the examination of the locomotor system *complete, it is*

now possible to outline with the physiotherapist a rational plan of treatment:

MANAGEMENT OF THE FIRST STAGE OF THE RECOVERY PERIOD.

Treatment is concentrated at first on two features:

(a) *the restoration of not only full but free range of active and passive movement*, by the methods already indicated (gentle stretch, hot packs, etc.) by slings, and by pool treatment if available

(b) *the encouragement of the weaker muscles.* In the first stage of recovery it is much more important to elicit even a weak contraction in muscles charted 0 or 1 than to raise — as is very much easier to do — muscles from grade 3 to grade 4. If the patient's attention is not concentrated on the weaker muscles their action tends to be overborne by their stronger neighbours, whether synergists or antagonists, and their residual, potentially useful, innervation may be wasted. The explanation lies probably in (i) the natural tendency of the patient's interest to focus on muscles which by their contraction release more proprioceptive impulses than their weaker neighbours, and in (ii) the increasing mechanical disadvantage under which the returning power of the stronger muscles forces the weaker to work. It is therefore important that in the early part of the recovery period, block or mass contraction of muscles which have different strengths is not allowed [illegible]

stimulated and exercised.

The routine of the physiotherapist at each session at this stage is the following:

(a) to ensure that the limb is warm. Neither joint movement nor muscle contraction is optimal if the limb is cold, very weak muscles will, in fact often fail to show any contraction unless the part is thoroughly heated beforehand. Massaging the limb may give sufficient warmth, or hot packs may still be necessary Better still, for one session daily the heat may be obtained by exercising the patient in the heated pool or (if a child) in the ward bath.

(b) the joints of the limb to be exercised are put through an increasing range of movement to obtain as free movement as possible, and to obtain just a little more stretch of any resistant muscle than was obtained on the previous day.

(c) the individual muscles are exercised systematically. *Weaker muscles* are exercised in the position most favourable for their

contraction — usually a position in which there is only moderate stretch and in which there is no resistance to the movement the muscle normally performs. Only one or two contractions per session may be obtained at first, after which the patient will say the muscle has tired. As the days pass more and brisker contractions develop until the contraction is well defined and does not disappear on repetition. Even quite young children will take an intense interest in developing, e.g. a "wrinkle" by contraction of the quadriceps at the knee, or in making the tendon of the tibialis anterior jump up on request. The *stronger muscles* are exercised to develop brisk, co-ordinated, free and repeated contraction, acting through the whole range of movement. Exercises to develop power are postponed until this has been achieved. In the absence of pool treatment, or in the intervals between sessions, slings are provided to reduce the effort required from the patient or from the muscle. Fatigue is not allowed and a falling off in performance is regarded as a definite indication for stopping an exercise.

(d) At the end of treatment the paralysed parts are left supported either in the position most favourable to maintenance of the newly-won limit of joint movement, or in the posture recommended for the acute stage, or in a position favourable to exercise. Considerable thought should be given to this selection of posture at the end of treatment.

If a limb is severely paralysed it is usually more profitable to divide the physiotherapist's treatment into 3 or 4 shorter sessions rather than into one or two longer ones in the day, particularly if the paralysis is severe enough to make the limb totally useless in between treatments. When it becomes possible, as a result of increasing power of the muscles themselves or by the help given by slings and counterbalancing weights, for exercises to be performed without the presence of physiotherapist or occupational therapist, the frequency of the seessions can be reduced.

THE HEATED POOL

"Pool treatment" has a special place in the treatment of cases of poliomyelitis, and particularly in the treatment of the more severe cases, and no discussion of physiotherapy in this condition would be complete without an account of its use. The heat of the water favours both the relaxation of muscles resistant to stretch and the voluntary contraction of all muscles. Its buoyancy assists the posturisation of the patient and, by taking part of the weight of



Fig 20 *Pool treatment* At the narrow end of the pool a boy is doing assisted active movements of his lower limbs At the wider end an adult is standing in the deepest of the three walking depths where she is supported by water up to mid-thorax

Note (*a*) Inflated 'ducks' for support of limbs exercising on the surface
(*b*) Sling to support patient's head when lying

limb or trunk muscles, possible active exercises which cannot be performed on dry land. [illegible] to carry the whole weight [illegible] standing, or walking, in the water [illegible] systems which are [illegible] on dry land.

The water is kept at a temperature of a warm bath (98°F), sufficient to prevent the patient feeling cold, however immobile his paralysis has rendered him. The [illegible] conditions are [illegible], and the local public health authorities take samples at regular intervals. Neither [illegible] nor those with [illegible] are allowed in the pool. [illegible] patients should not use the pool for at least [illegible] from onset.

The pool should be long enough to allow the patient to walk six or seven steps without turning, and have adjustable hand rails and graded depths to allow exercises in the lying, sitting and standing positions for all heights of patient. A head sling and inflated rubber rings are used to support the head, trunk and limbs as required.

Patients with severe respiratory paralysis must be watched carefully in the pool, as the weight of the water on their chest can seriously interfere with their respiration. A rubber ring to support the chest out of the water and flexion of the head in relation to the chest so as not to interfere with accessory sternomastoid action will help them to tolerate pool treatment. Particular care must of course be taken to ensure that they run no risk of catching a cold, since any respiratory infection can severely set back their recovery programme. One pool session a day for 5 days a week is the normal routine. At first the session is short (about five minutes) but later the patient can tolerate 30-45 minutes.

Physiotherapy in the pool does not differ markedly from that on dry land. As already mentioned posturisation is easier and the assistance given by the buoyancy of the water makes everything less tiring, so that a great deal more can be done in the time available and it is usually possible to work through a set routine of exercises of all parts of the body without exhausting the patient. The need to provide the patient with a fixed point to obtain true movement is increased in water; thus the pelvis must be held for exercises of the muscles of the hips, the thigh for exercises of the knee, and the scapula for exercises of the arm. If true movement is not insisted on, and the patient is allowed to obtain his movement by wriggling, his performance on land will fall disappointingly behind his performance in water.

Sitting and standing in the pool [illegible]

morale. If recumbency has been prolonged dizziness (apparently of vascular origin) and true vertigo (associated with nystagmus) are to be expected. The vertigo is best corrected first by accustoming the patient gradually to sitting and lateral turning in bed — this may take several weeks. The dizziness is often associated with poor abdominal musculature and is reduced by preliminary application of an abdominal binder. In fact, it is a wise precaution to prescribe an abdominal binder for use on sitting and standing to all cases with weakness of the abdominal muscles. The support given increases the patient's tolerance of these positions and prevents stretching of the abdominal muscles. If the abdominal weakness persists a more permanent support in the form of either a strong corset or a Salz belt is required (see p. 124).

Walking in the pool is usually started at the deepest level suitable for the patient's height — ideally the water should come about half-way up the patient's chest. If the water is higher the buoyancy hinders walking since the patient's lower limbs tend to float up, particularly if the gluteal and trunk muscles are weak; if it is lower less support is given to the trunk; if the water reaches only to below the hips it is usually more hindrance than help in walking, since the assistance obtained from its support of the weight of the trunk is gone and is replaced by the resistance to the thighs at the surface. The importance of securing a correct standing posture (in adults with the aid of a mirror) before starting walking is as great or greater than in walking on dry land.

The amount of exercise to be performed whether on land or in the water is determined by the patient's performance. As soon as the performance deteriorates the exercise is interrupted (see p. 88). On the other hand, if the exercise can be performed perfectly and without deterioration the need for it is passing and the patient should progress to the next stage. Thus, when the patient can walk up and down the pool length as many as eight times without deterioration in his performance, he should begin to graduate to a shallower depth or — if already in the shallowest depth suitable to his height — to walking on land. The final use of the pool in the severely paralysed is to teach control in stepping from one level to another.

MANAGEMENT OF THE SECOND STAGE OF THE RECOVERY PERIOD.

The first stage of treatment, in which concentration is mainly on the weak muscles and on obtaining brisk free active and passive movement, rather than on power, merges into the second stage in which more vigorous treatment is adopted. No specific time for the beginning or end of these stages is suggested because, as mentioned elsewhere, the actual number of weeks that each stage lasts varies widely from patient to patient — and also from limb to limb in any one patient. Suffice it to say that the second stage is reached when passive movement is free, the contraction of the muscles is "brisk" — irrespective of their power — and it is felt that the residual innervated motor units of even the weakest muscles have been given ample opportunity to show their contractility One's judgement in this matter is, of course, influenced by the distribution and severity of the paralysis; doctor, physiotherapist and patient are all more ready to devote time to a very weak muscle if it is one of very few muscles with residual innervation, or has particular functional importance

By now the muscles will fall naturally into two categories:

(1) those which are nearly or quite adequate to their task, but require training to bring them to their full power,

(2) those which are too weak to fulfill the function required of them.

The former must be developed by exercises to develop both maximum power and maximum skill in their use. The latter must be replaced by substitution, viz. by other muscles (the acquisition of skill and development of trick movements), by instrumentation to give either stability or spring movement, or by surgical reconstruction.

Training of muscle for strength. It is a familiar observation, in athletics and in every physiotherapy department that the strength [illegible] [illegible] way (progressive resistance exercises) is associated with an increase in the size of the individual fibres of the muscles taking part in the exercises. Other changes which it is reasonable to believe might also occur are. an increased vascularity of the muscle, an increase in the vascularity of the related part of the spinal cord — or even of the cerebral cortex — and changes in the nerve cells and axons

concerned. At present, however, work on this subject is incomplete and it is better to confine the discussion simply to the practice of training of muscles to increase their strength.

The essential feature of all effective training routines applied to normal or partially denervated muscles appears to be the adjustment of the task set to the muscle so that, although within its capacity, it is always just ahead of its easy performance. De Lorme's progressive resistance exercises embody this principle (⁸⁶); their effect is probably to induce a maximal contraction of the muscle, with the result that the load cannot be distributed by alternation of activity amongst the available motor units and no motor unit remains unstimulated. The relative failure of submaximal contractions to stimulate the development of hypertrophy and increased power may result from the fact that some motor units remain always as a reserve which is not used. Exercise to fatigue is a poor stimulant, presumably because as soon as fatigue begins either only submaximal contractions are obtained, or the action of the muscle for which the exercise was designed is replaced by the action of other muscles.

An interesting feature of most training routines applied both to normal muscle (as in professional weight-lifting) and to partially denervated muscle — is the small number of maximal contractions which are required to stimulate the development of muscle power. Ten to thirty maximal contractions a day, three times a week, appear to be as effective as — or on occasions more effective than — a greater number of contractions or a greater number of sessions. In our experience thirty maximal contractions maintained for not more than 3-5 seconds, at an interval of half to one minute, are an effective stimulant to the development of power in a selected muscle. The relatively long interval between contractions is required to prevent falling off in performance (and consequently submaximal contraction) at the end of the session.

In poliomyelitis careful selection of muscles to be trained is essential, since unselective development of all residual innervated muscle can be harmful by exaggerating an unfavourable muscle imbalance or by overloading muscles on whose contraction the muscle under training depends for its stability. Thus, a weight-lifting exercise for the biceps or triceps can on occasion do harm by causing excessive pull on the stabilisers of the shoulder whose grading is lower than that of the larger muscles; with each contraction the scapula tilts or slides out to the axilla and the small stab-

lising muscles, e.g. the rhomboids and the levator scapulae become so stretched that their function is permanently reduced. This mutual independence of muscles of varying sizes and strengths often makes it difficult to devise suitable resistance exercises for poliomyelitis muscles. The difficulty can usually be overcome *either* by a preliminary training of the stabilisers before any attempt is made to train the muscles dependent on them, *or* by designing the exercises so that maximal contraction occurs in all these muscles but no actual movement takes place (static contraction). Static and dynamic contractions appear to be equally effective in training muscle power, once free movement at the joint has been satisfactorily developed. If the power increase obtained is measured as a dynamic contraction (e.g. by weight-lifting) the curve always appears steeper than if the static power is measured — but the same dynamic and static curves will be obtained during training whether the training is actually performed by dynamic or by static contractions. The difficulty with static contractions, is usually to devise a system of progressively increasing the load, however, in the absence of equipment such as an accurate strain guage dynamometer, it appears to be satisfactory simply to demand a series of *maximal* contractions; these can be obtained by asking a patient to try to his utmost to move an object which is, in fact, immovable. Where the problem of weak stabilisers does not arise, simple weight-lifting of progressively increasing weights is suitable for muscles over grade 3, for muscles below grade 3 the same effect is produced by gradually *decreasing* weights adjusted to help the muscle.

FATIGUE

A word should, perhaps, be said on fatigue. The term is used here to describe *deterioration of function during repetition of an exercise*. At least two distinct types of fatigue must be recognised. Firstly, there is the fatigue of a muscle by voluntary exercise of a simple movement for which it is almost entirely responsible, concentration being kept entirely on the movement, e.g. exercising the biceps or triceps by repeated weight-lifting until the contractions are no longer maximal and the subject can no longer lift the weight to the same height (though he can still lift a smaller weight). Secondly, there is the more generalised fatigue induced in a patient performing a more complicated functional movement, such as walking. Here, as the weak muscles tire and their function deterior-

ates, other muscles take over the exercise, which continues, not as fatigue of the weaker muscles, but as exercise and fatigue of other muscles not usually taking part in the movement. Both types of fatigue are undesirable, the former because by replacing maximal with submaximal contraction it renders the exercise less effective: the latter because only too often it results in the development of faulty postures and gait.

It is unwise to exhort patients to perform complicated movements, such as walking, sitting or standing, to the limit of fatigue. A common example is that of the patient with weak abductors of the thigh who can walk a few paces with perfect posture and perfect gait, but as he tires develops an abnormal gait, even to the extent of a positive Trendelenberg sign. In such a case the actual exercise of walking must be discontinued the moment deterioration or the slightest sign of fatigue is detected Over the same period specific exercises are given to develop the power of the muscles which have been found *to be inadequate to their task*. If this is done correct posture and gait are maintained and over the weeks it will be found that the number of steps which can be taken will be steadily increased. The physiotherapist's job is, therefore, not to urge on her patient greater distances or longer times spent sitting or walking Instead she should prevent the patient from exercising beyond his limit and at the same time detect which muscle of muscles are creating the limit and devise for them exercises which will effectively develop their potential power, so that they become adequate to *their task*.

ELECTROTHERAPY

One is occasionally asked whether there is a place for electrical stimulation in the treatment of muscle paralysed by poliomyelitis Theoretically, there seems little purpose in stimulating the innervated muscle fibres, since as good or better contraction can usually be obtained by voluntary stimulation. There seems equally little purpose in stimulating the denervated fibres, since their denervation *is* presumably permanent, and they *will not be taking* part in voluntary contractions. On the other hand, it can be argued that the contractions in the denervated muscle increase its suppleness and reduce the mechanical resistance offered to the voluntary contraction of the residual innervated fibres.

In practice, electrical stimulation does occasionally seem to improve the voluntary contraction of a muscle, but it is usually

difficult to say whether the improvement is obtained simply by increasing the patient's awareness of the muscle, or by a more direct effect on the muscle itself. The benefit is usually obtained in a few sessions and prolonged courses of electrotherapy are rarely indicated, less perhaps because the treatment is entirely valueless than because it appears to have less value than other forms of treatment with which the patient's day is already fully occupied.

RECOVERY FROM RESPIRATORY COMPLICATIONS — WEANING FROM RESPIRATORY AID

PARALYSIS OF SWALLOWING

Paralysis of swallowing, like paralysis of micturition, is rarely, if ever, a permanent complication of poliomyelitis, and the measures required to compensate for it can usually be discontinued sometime within the first three weeks—provided precautions are still taken to deal with any secondary pathological process which has developed in the lungs. Minor weaknesses may, however, persist longer, and perhaps indefinitely. The optimum position for swallowing in such cases depends on the site of the weakness. Usually a sitting position of 70° in which gravity assists the bolus to fall posteriorly is the most comfortable. Only one lateral food channel may be used, and this may mean that the swallowing is easier when the head is turned away from that side. These minor weaknesses are usually little or no disability so long as the patient is healthy; but there may be risk of pooling in the pharynx and aspiration when the normal control is weakened, e.g. by debilitating disease or anaesthesia, and it is sometimes wise to inform the patient or his relatives or doctor of the significance of the finding.

PARALYSIS OF RESPIRATION

Paralysis of respiration on the other hand, is often associated with considerable permanent damage to the respiratory motoneurons Its recovery, therefore, follows the same pattern as recovery from other spinal paralyses, and consists of the four components already described — viz. true recovery, release from inhibition, training of residual motor units and adaptation. Two additional factors also have considerable effect in the immediate post-acute stage, these are (i) the resolution of residual pulmonary [illegible]

level, with consequent increase in the oxygen-carrying capacity of the blood.

In practice, cases with respiratory paralysis fall into two groups:

The first — and largest — group consists of cases who recover respiratory independence fairly rapidly, e.g. within three months of onset, and suffer little permanent inconvenience apart from loss of coughing power which may demand more than usual care in dealing with winter respiratory infections

The second—and smaller—group consists of cases with extensive permanent damage to their respiratory motoneurons. Weaning from respiratory aid may take anything up to two years but is usually complete in the end. In a very small "hard core" of cases (older subjects and those with virtually total paralysis and cases with severe residual pulmonary pathology, aggravated perhaps by a tracheostome) respiratory aid by Bragg-Paul, cuirass or rocking bed may be required at night indefinitely.

It is not possible to determine from any single test, (e g the vital capacity, the chest expansion, analysis of the action of the respiratory muscles) whether or not a patient should be able to manage without respiratory aid The only sure test is to try whether or not he can, in fact, learn gradually to do so Some of the factors concerned may be quoted· (1) the rigidity or elasticity of the chest-wall, (2) the strength of the expiratory as well as of the inspiratory muscles, (3) the proportion of the respiration carried out by the diaphragm, the intercostals and the "accessory" respiratory muscles, (this is important in determining whether the patient can *sleep* without aid) (4) the restriction of the movement of the diaphragm by wind or constipation, (5) the condition of the lungs, (6) the efficiency of the cardiovascular system, (7) the haemoglobin content of the blood, (8) the patient's metabolic rate. The role of all these factors must be recognised so that the optimum conditions for weaning can obtain In particular, two principles should be recognised·

Firstly, the respiratory muscles cannot be "trained" by allowing them to work to exhaustion, since the anoxemia which their own fatigue causes leads to rapid deterioration of the conditions under which they have to work, with the result that the period of recovery from fatigue is unnecessarily prolonged.

Secondly, weaning from respiratory aid should have priority ıver locomotor recovery: otherwise the patient's activities may utstrip his respiratory capacity, so that, in the intervals between :tivity he comes to rely more and more instead of less and less on ry aid, and the need to compensate by the characteristically) of respiratory aid at night for the increasing burdens put

upon the respiratory muscles during the day seriously interferes with weaning from such aid. In fact, failure to observe this cardinal rule of priority of respiratory over locomotor development leads to such anomalies as patients walking about, perhaps running a home or going to work in the daytime, yet dependent upon respiratory aid when they flop exhausted into bed at night. In such cases successful weaning can only be achieved by temporarily drastically reducing their daytime activities and only increasing them again when the respiratory capacity is again in the lead. It is interesting that once respiratory independence is achieved it is rarely — if ever — lost again unless cardiovascular or pulmonary disturbances supervene. It is therefore well worth the extra time and trouble spent in securing it. It is highly valued by the patient.

WEANING METHODS

At first when the cabinet respirator is opened the patient may not tolerate more than half a minute without some substitute aid. This can be given either by manual compression of the chest (14-20 per minute) or by positive pressure through face mask or mouthpiece. Gradually the time for which he can be left without [illegible]

need warrants it. Instead, aid should be offered as soon as [illegible]tory function appears to be inadequate, preferably before the patient becomes aware of his failure. Intermittent assistance with a few deep breaths will often considerably prolong the period for which a patient can stay out of the lung at any one time and so speed his progress and improve his morale.

By the time the patient can tolerate unassisted breathing for as long as 30 minutes it is useful to have a comfortable bed, which the patient knows is for him, in sight: it is left to the patient to suggest when he should be transferred to it, first for short and gradually for longer periods. But still he is returned to the lung before he shows acute distress. Early signs of distress are reluctance to talk, increased activity of the accessory respiratory muscles and sweating. A tendency to sweat and to fall into a deep sleep as soon as the respiratory aid is restored is definite indication that the pace of weaning is too fast and should be slowed: the patient should be reassured that even a few whole days' rest in the respirator will not

[illegible]

to the patient in advance and he should, if possible, be the one to suggest that he is ready for it. In this way the patient feels himself to be the keenest member of the weaning team and his co-operation and confidence are never lost It is obvious that measures which "cheat" the patient — e.g. opening a respirator arm-port or reducing the pressure without warning — are never advisable; not only do they lose the patient's confidence in his attendants but also they ask him to breathe in adverse circumstances, i e. with his accessory muscles cramped by the neck collar and even against the motor's reduced action.

Occasionally difficulties arise, as already mentioned, from over-ventilation by the respirator (83) The consequent low carbon dioxide level leaves the patient without the normal stimulus (carbon dioxide retention) to breathe. When he is taken out of the respirator he may become very uncomfortable and distinctly cyanosed, but show no attempt to use the accessory muscles to increase the ventilation, though these may be known to be innervated In such cases the venous blood will show low carbon dioxide levels and weaning will be favoured by gradually reducing the excessive artificial ventilation and by increasing the frequency of freedom from aid

The final stage of the weaning process is learning to sleep without aid Generally speaking, patients who have had assistance for sleeping for many months or even years are more difficult to wean from respiratory aid than those with only short experience of such aid, even though their vital capacities are similar This would seem to be caused partly by their becoming accustomed to the very deep sleep usually associated with such aids, and partly by the tendency of most aids to cause overbreathing with resulting adaptation to low carbon dioxide levels. For his first "nights out" the patient should be warned that his sleep will be lighter (and in that sense more normal) than in the respirator and that he may find that he wakes often in order to increase ventilation with his accessory muscles. He should also be assured (1) that the loss of sleep, though it may be boring, will not do him any harm, (2) that there is absolut[illegible]

wakir [illegible]

relati [illegible]

the bedside for the first two or three unassisted sleeps. Hypnotics are of course contra-indicated, when a patient is to try to sleep without aid. Occasionally benadryl may help the patient to settle calmly. The benadryl seems to be useful less as a sleeping drug than as a relaxant. Before spending a night without aid the patient should have had a restful day and so start the night without carbon dioxide retention and with his respiratory muscles working under optimal conditions. At first a regime of, for example, one night without, three nights with aid, is observed, the day before a night out being particularly restful. The proportion of nights out: nights in is gradually reversed within the patient's tolerance until he himself feels that he can sleep well enough unaided. The respirator should not be finally removed from his sight until he is anxious to see it go!

Wherever possible the process of weaning should be direct from tank respiration to unaided respiration, and the temptation to ring the changes on a variety of apparatus should be resisted. Each type of apparatus requires a certain amount of "getting used to" and it is only too easy to waste the time and skill which should be spent on weaning from all apparatus on getting a patient used to some substitute aid. It is doubtful whether any of the apparatus so far designed really assists in *training* the respiratory muscles; most tend not only to take over too much of the respiratory function (since they control both rate and depth of respiration) but also often actually interfere with the spontaneous respiration. The exception is the positive pressure belt, where the aid is given only to expiration and the depth of inspiration beyond that of elastic recoil is left to the patient's own respiratory centre and muscles. This overriding by apparatus of the normal physiological control of rhytm and depth of respiration is a striking feature in patients using respiratory aids; they themselves will sometimes volunteer that it may take them several hours after leaving the respirator to cease to breathe in the pattern it imposes on them. Consequently, it is important not to let weeks and months go by while the patient merely learns how to use different kinds of aid.

If, however, the need for respiratory aid is likely to be prolonged it may well be worth changing fairly early and completely to, for example, a comfortable cuirass which will allow the patient to lie on an ordinary bed, to become accustomed to sitting up, and to have physiotherapy to arms and legs without discontinuing the aid. The change should be gradual over one to two weeks to allow for

adaptation to the new forces exerted on the trunk and to avoid pressure sores both directly from the cuirass and its straps and indirectly from counter-pressure on the mattress. The line of seal should be treated as a pressure point in the same way as other pressure areas. The patient should have a layer of cloth, gamgee tissue or multopren between him and his cuirass.

Some patients prefer the rocking bed, since on it they are able to breathe completely unencumbered. An occasional patient will be able to sleep on it all night

Patients welcome reductions in the apparatus attached to themselves and are good witnesses as to whether they are getting sufficient ventilation from new apparatus. If the new apparatus is unsatisfactory it is more often the apparatus than the patient that is at fault! It is preferable to avoid combining a change of hospital with a change of apparatus; a change of either alone can worry a patient considerably; a change of both together may prove a harrowing — and even an unsuccessful — experiment It can be

to its parent hospital later

Weaning from positive pressure respiration administered through a tracheostome and the final closure of the stoma presents a few additional problems. Each of the functions of the apparatus and stoma must be dispensed with in succession·

1. The need for the cuff to be inflated to ensure that foreign material does not enter the respiratory tract from the trachea is removed fairly early by the natural recovery of the muscles of swallowing Inflation of the cuff is now needed only to prevent leakage of pressure into the pharynx when the pump is in use, and when it is not in use the cuff can be deflated: this enables the patient to talk.

2. The need for the positive pressure pump as a means of artificial respiration is removed, either by direct weaning from the pump to spontaneous respiration, or, where recovery of the respiratory muscles is insufficient, to some other form of assisted respiration which does not require the tracheostome. A cuirass respirator will usually give sufficient help at this stage and the patient should become accustomed to it within one to two

3 The need for humidification may persist, after the

discontinued, to prevent secretions from becoming "sticky" for lack of the humidification and warmth previously supplied with the positive pressure respiration. It may be necessary, therefore, to supplement respiration by the cuirass with short spells on the positive pressure pump for the sake of humidification. Gradually the need for these spells is reduced with the help of a humid atmosphere in the room combined with a scarf loosely tied in front of the tube to form a local warm damp pool of air.

4. When both the positive pressure pump and humidification are no longer required the cuffed tube can be replaced by a plain one, since by now the sole function of the tracheostome is to allow suction of the secretions in the trachea.

The final stage of closure of the tracheostome is now in sight. Various criteria for *when* it should be safe to close a tracheostome in these patients have been suggested, e.g. when both diaphragms are working, or a certain minimum vital capacity is reached. In practice, however, these requirements would condemn unnecessarily a number of patients to a permanent stoma; the most useful criterion is simply that secretions are minimal and can largely be controlled by posture and physiotherapy, and the patient's own coughing and that there is an alternative respiratory aid available. It is an advantage also if the patient can breathe spontaneously for short periods in the prone position. So long as the tube remains in situ some suction will continue to be required, since the tube both increases secretion and interferes with the normal route of its removal. It is therefore futile to wait for complete absence of secretions.

The obvious preliminary stage of closure of the stoma would seem to be to diminish its size by reducing the size of the tube in use. In practice, however, the mechanical trauma of too loose a tube in the trachea may increase the secretions (this trauma can be considerable if the sternomastoids are overactive) and it may therefore prove easier to dispense with these stages and pass straight to strapping over the stoma as securely as possible. In some cases a silver cannula, which is less irritant than rubber tubing, may give a useful intermediate stage.

During the process of closure every precaution should be taken to ensure that secretions are not allowed to accumulate in the lungs and that intercurrent infection does not occur. These precautions include.

(1) physiotherapy to the chest and postural drainage (head-down

tipping all night and every 2 hours in the day is a worth while precaution in cases with impaired cough),

(2) antibiotics to prevent infection with increase in secretions,

(3) protection from infections and respiratory disorders (an influenza vaccine is a wise precaution if closure is to take place during the influenza season),

(4) all facilities available for re-opening of the stoma, suction and bronchoscopy, etc.

Once closure is completed the patient should present no more problems than other cases of paralysis with respiratory weakness, and weaning from respiratory aid, if not already complete, should continue along routine lines — allowing for a temporary setback [illegible]

[illegible] solution containing adrenaline: the skin edges round the stoma are freed and closure is obtained *in two layers if possible* Effective continuous suction in the wound is required throughout the operation to ensure that neither blood nor anaesthetic solution are aspirated. Twenty-four to 48 hours' post-operative postural head-down drainage (either intermittent or continuous) is recommended as a routine precaution for those with inadequate coughing power.

Finally, a word should be said about *the management of respiratory infections in patients whom respiratory paralysis has left with impaired coughing power and limited chest expansion.* A "cold", or, worse, an attack of "influenza" can be a serious setback to recovery and can even be fatal if not managed correctly. Prophylaxis is, of course, of prime importance: visitors, nurses, physiotherapists and doctors must conscientiously observe the rule of "keep away or wear a mask" when they have a cold. It is worth while administering influenza vaccine in the autumn and again around Christmas time to all cases in whom intercurrent respiratory infection is to be feared.

Should the patient suspect "a cold", antibiotic therapy is valuable for preventing secondary infection, and routine postural drainage (raising the foot of the bed, and prone turning with assisted coughing at regular intervals) is instituted at the slightest suggestion of increased secretion. Children without effective cough should be tipped in bed head-down in the prone position for about an hour

and then supported for a few minutes over the side of the bed to cough out secretions (see fig. 21). Their parents should be taught the principles and practice of this drill before they leave hospital. Expectorants are absolutely contra-indicated and the contents of "cough mixtures" must be checked from this point of view before they are administered. It is surprising how effective expectorants thought to be "mild" can show themselves to be in a patient who cannot cough up his secretions. If secretions do develop, the administration of an antihistamine which is a strong bronchial dilator, or trypsin, by aerosol, combined with steep head-down postural drainage, and physiotherapy as suggested in the acute stage help to ward off atelectasis. Finally, should atelectasis occur aspiration under direct bronchoscopic vision (with a short-acting anaesthesia such as pentothal) will usually result in considerable improvement. Usually the first "cold" is the worst; as respiratory function improves and the patient learns the importance of early treatment the danger steadily decreases. The exceptions are cases with progressive scoliosis in whom the respiratory impairment will tend, if anything, to increase.



Fig 21 Position for coughing in a child with impaired coughing power The firm support under shoulder and pelvis gives a paralysed child a sense of security

CHAPTER 15

THE MANAGEMENT OF PERMANENT DISABILITY

However good the treatment in the acute and the recovery stages and however courageous and determined the patient and his relatives, irreversible damage to a significant number of motor nerve cells must cause some degree of permanent disability, the degree being determined by the distribution and number of cells destroyed. Probably about half of the notified paralytic cases of poliomyelitis are, if they survive, left with a disability which ranges from relatively minor dysfunction to complete helplessness. The management of these cases and the reduction of the disability to the absolute minimum compatible with their residual innervation forms an increasing proportion of the work of the orthopaedist as each year adds its quota of cases to the accumulated total of previous years. In the U.S A. it has been calculated that (excluding amputations) poliomyelitis cases make up 30 % of all orthopaedic patients with disablement under the age of twenty-five (83).

To discuss the management of the permanent paralysis in a detail comparable with that used so far to discuss the management of the acute and recovery stages would be to write a large part of an orthopaedic textbook. The discussion would have to cover such a wide range of subjects as · the management of abnormalities of the foot; the problems of deformity of the spine; the whole field of reconstructive surgery (including the principles and methods of tendon transplantation, arthrodesis, osteotomy and the management of unequal length of the limbs); the making and fitting of orth[illegible]

para[illegible]

boo[illegible]

give[illegible]

were ignored altogether, and the reader were simply referred to the standard text-books of orthopaedics. It has therefore seemed reasonable to try to show quite briefly, for the benefit of the increasing number of people from diverse branches of medicine and social welfare who nowadays come into contact with the enlarging population of poliomyelitis cases, some of the methods used by the orthopaedic and auxiliary services to prevent potential deformity and to help to compensate for the permanent paralysis.

The management of adult and child cases is slightly different. In

adults emphasis can be freely laid on restoration of function, and reconstructive surgery can be used as soon as it is felt that maximum recovery has been reached. In children, on the other hand, the emphasis is less on reconstruction than on the protection of the paralysed parts over the period of growth, so that the disability of bone and joint deformity is not superimposed on the paralysis. The need for constant and *expert* supervision of children, even with relatively mild paralysis, throughout the period of growth cannot be overstressed. Severe cases, in whom the need for such supervision is obvious, are less likely to slip through the administrative net than those with relatively mild paralysis. These are often sent home from a fever hospital with the apparently reasonable expectation that the already rapid recovery will be complete in the next few weeks. If this expectation is not fulfilled, there tends to be delay in seeking advice and the patient may not be seen before deformity has already started to develop. It is important to realise that it is impossible — however great one's experience — to forecast whether all muscles will recover completely or even equally and that it is therefore essential that all cases which have shown paralysis, however mild, in the acute stage, should be given routinely an out-patient appointment at an orthopaedic clinic for 8—12 weeks after onset to determine whether or not further supervision is required; in this way any deforming tendency due to muscle imbalance will receive regular skilled orthopaedic supervision from its earliest and most controllable stage.

Apart from this important *preventive* aspect of the management of permanent paralysis, measures to reduce disablement fall into three main categories: apparatus, surgery and rehabilitation.

1. *Apparatus.* The apparatus required in cases of poliomyelitis ranges from small, often "lively" splints, designed to assist or replace normal movement, to calipers and spinal supports designed to give stability or to control the development of deformity. It also includes a wide range of apparatus and gadgets used by, rather than fitted to, the patient, ranging again from small gadgets such as special handgrips for use in a paralysed hand to overhead suspension apparatus for the arms, walking machines, wheelchairs, (propelled by the patient or by motor or electricity), and devices for the hand control of cars. Some of this apparatus is prescribed to tide the patient over a long recovery period, with a view to its discard at a later date: some will be of permanent use.

2 *Surgery.* The surgery of poliomyelitis is often classified as corrective and reconstructive, but in practice one operation often serves both functions. A few of the commoner operative measures are. transplant of the origin or insertion of a muscle in order either to correct deformity or to transfer the power of a still innervated muscle to pull on a more necessary tendon; osteotomy of a bone to increase mechanical advantage, restore stability or correct deformity of abnormal bone growth; and fixation of a joint in which stability is more important than movement. One, or a series, of such operations may so improve function as to enable the patient to dispense with cumbersome apparatus, or actually to recover [illegible]

[illegible]

where sensation is impaired. The time at which operation becomes justified is often discussed, rather unsuitably, in general terms, i e. "at the end of six months", or "not until the end of two years." In fact the time varies according to the state of recovery, not only of the muscles particularly concerned, but of other muscles only indirectly concerned, and it is difficult to make generalisations. Two important factors are the extent of the paralysis and the age of the patient.

3 *Rehabilitation.* This term must be used in its widest context, to cover *inter alia* the provision of suitable home surroundings (with a view to reduction not only of the difficulties of the patient, but also of the burden on his family), the selection and provision of educational facilities most suitable to a particular child, and either the modification of a patient's original employment to suit his reduced capacity, or the selection of suitable alternative employment and the provision of the necessary training for it

APPARATUS AND SURGERY

The application of the first two groups of aid, apparatus and surgery, is best considered from the point of view of the part affected.

PARALYSIS OF THE MUSCLES OF THE UPPER LIMB

It has already been mentioned that paralysis in the upper limb is usually most severe in the proximal muscles, and that even with severe involvement there is often some sparing of the long muscles

of wrist and hand. In severe cases, therefore, one is often concerned with overcoming the proximal disability in order to allow maximum use of the residual power in the hand. While the patient is in bed an overhead support, on which the arm, forearm and hand can be slung, allows any residual power to be used unhampered by gravity, brings the hands within useful range of eyes and mouth, and counteracts the tendency to adduction contracture of the shoulder (see fig. 22). In this position the patient can exercise continuously — without help from nurse, physiotherapist or occupational therapist — and can learn to write, turn pages and feed himself before any muscles of the upper limb have reached Grade 3 (anti-gravity). The effect on morale after weeks or perhaps months of inactivity is obvious.

In the ambulant patient with paralysed shoulder muscles an abduction splint should be worn for as long as the tendency to adduction contracture of the shoulder persists. It can then be abandoned unless the patient feels the weight of the limb on the shoulder girdle uncomfortable, or unless he finds — as may be the case if both shoulders are affected — that it is useful to have one hand in the raised position. Later, if the muscles that insert on the scapula are strong, arthrodesis of the shoulder joint in 60° scapulo-humeral abduction can give a useful active movement, at the expense, however, of substantially losing internal and external rotation. Arthrodesis of one shoulder finds its main indication in cases with severe bilateral paralysis.

Weakness of flexion of the elbow in the ambulant patient can at first be corrected by a sling to bring the hand into a useful position. If the triceps is strong a flexion spring can be included to give the effect of active flexion and extension (see fig. 23).

Permanent impairment of active elbow flexion can best be corrected by transplant of the triceps or pectoralis major into the biceps but if these muscles have been too severely affected, either a muscle slide of the proximal origin of the long extensors or flexors of the wrist and digits, or a bone block to prevent full extension, may have to be considered.

Weakness of the long muscles of forearm and hand requires correction according to the particular combination of paralysis, some form of splintage which stabilises the wrist in about 10° dorsiflexion and allows active movement of the digits by carefully balanced live splinting is useful in cases where weakness is severe. Loss of opposition of the thumb, which may occur as an isolated paralysis of the

Fig 22.



Fig 23 Spring support for weak biceps muscle

Fig 22 Overhead suspension and slings enabling a patient with inadequate shoulder muscles to use the less affected forearm and hand muscles. Note also the 'polly perch' which enables a patient with good arms to move about in bed.

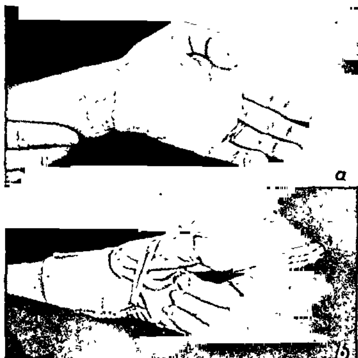


Fig. 24 Glove splint for paralysis of the thenar muscles, enabling the patient to use residual power in the long flexor muscles of thumb and fingers.

(*a*) 'Simian' position of thumb and wasting of thenar eminence

(*b*) Fingers-thumb pincer action with thumb in abduction-opposition.



Fig 25 'Unicorn' page turner for a patient with flail arms.

thenar muscles or as a part of more extensive damage, is a particularly disabling paralysis and also requires live splinting, for which a [illegible]

plantation to distribute the available muscle power to its best advantage, supplemented, where power is insufficient to move all the joints, by arthrodesis of those in which stability is more useful than weak movement, notably the wrist joint. An alternative arrangement, for use when very little power remains in the long muscles of the forearm, is to use active dorsiflexion at the wrist to give passive palmar flexion of the digits and opposition of the thumb by the resulting pull on shortened tendons crossing the flexor surface. The possibilities of tendon transplant in wrist and hand make it important that every ounce of strength be developed in the forearm muscles, even though their potential use may not be immediately obvious.

PARALYSIS OF MUSCLES OF THE TRUNK

Weakness of the spinal muscles. In children with weakness of the trunk muscles the importance of supervision by those familiar with the orthopaedic problems of spinal deformities from any cause should again be stressed, since deformity once initiated can develop very rapidly. In adults also considerable attention must be paid to even minor weakness of the trunk muscles so that they are not subjected to undue fatigue which can lead to faulty posture and chronic backache. Gradual increase in sitting, standing and walking will avoid deformity and usually obviates the need for cumbersome support of the spine in most adults A few cases, however, will eventually have to be fitted with a moulded leather or plastic spinal support if they are ever to learn to sit and walk again.

Weakness of the abdominal muscles should similarly not be ignored. The large area of unsupported musculature makes the abdominal muscles particularly susceptible to stretching so long as even moderate weakness persists, and even when their weakness is only mild these muscles need to be supported until sufficient tone is recovered. It is obviously not enough for the patient to produce a good contraction in the abdominal muscles for a few seconds while being examined, he must be able to maintain the contraction without fatigue throughout the period of sitting and standing and in

spite of distraction by the need for attention to other weak muscles. A many-tailed bandage gives sufficient support when the patient begins to sit up in bed, and reduces the feeling of faintness and weariness in the early stages of both sitting and standing. If weakness of the abdominal muscles persists, a Spencer corset — which also gives useful lumbar support — should be used, and this can be left off gradually, i e. for increasing lengths of time, as the muscles recover and are developed by exercises. An adult patient is usually himself the best adviser on his need to wear his corset. Special attention should be paid to the fitting of an abdominal support when there is respiratory impairment; respiratory cases usually benefit from a support which gives some counter-push to the diaphragm, but too high or too tight a support can seriously interfere with their respiration.

PARALYSIS OF THE LOWER LIMBS

When paralysis of the muscles of the lower limb is severe the disability is primarily one of loss of stability, chiefly at hip and knees and to a lesser extent at the ankle. The apparatus required to correct instability at hip and knee is a caliper in one or other of its variations, viz with bucket or ring top, with or without a pelvic band, with or without automatic knee joint, and with different variations at the ankle according to the particular distribution of the paralysis. Similarly, the apparatus required when paralysis is confined below the knee will vary according to the distribution of the paralysis; a double or single iron with T-strap will help to maintain normal alignment of the foot, and some form of toe-raising device or block will control drop-foot or the reverse calcaneus deformity.

Surgery for paralysis of the lower limbs forms a considerable part of orthopaedic practice Among the commoner operations are:

(a) shifting of the origin of shortened muscles of the hip to correct deformity — e.g lowering the upper insertion of the tensor fascia femoris and long head of rectus femoris in cases with hip flexion deformity.

(b) osteotomy at the lower end of the femur to give a "back-knee" stability; this operation, to be successful, requires either residual power in the gluteal muscles or some tightness of the calf muscles.

(c) lengthening of the tendo Achillis.

(d) arthrodesis for correction of foot posture.

(e) tendon transfers to control ankle and midtarsal movement, either alone or combined with (d), sometimes used prophylactically to prevent deformity in children.

Two further problems may arise in connection with paralysis of the lower limbs; namely vascular disturbances and inequality of growth of the lower limbs.

Vascular disturbances. The cold blue appearance of the limb in lower motor neuron paralysis is a common feature of poliomyelitis and in a few cases the vascular disturbance may prove a serious problem, causing considerable discomfort and, occasionally, as when chilblains develop and ulcerate, considerable loss of education or working time All cases with paralysis of the lower limbs should be given instruction on how to control the condition before leaving hospital, viz: two or three layers of warm socks, massage, contrast bathing and the local application of long-lasting rubefacients will often suffice. Occasionally, however, the condition proves intractable and sympathectomy is necessary: the results are usually satisfactory.

Unequal growth of the lower limbs. Relative shortening of the more affected limb can be a serious problem in children The relative importance of the different factors which cause diminished growth in a limb with lower motor neuron paralysis is not exactly understood, but one can say that the disparity in growth between the two limbs tends to be most marked when the disease is developed early, when there is a marked difference in involvement of the two limbs, and when the soft tissues are in poor condition (contractures and poor circulation)

Small disparities in length can be compensated by raising the shoe on the shortened side to maintain a level pelvis and so protect the hips and spine A raising of more than an inch, however, can be quite a disability, and a variety of procedures are in use for obtaining equalisation, all are somewhat uncertain in their effect. Growth can be accelerated on the shorter side by a bone block operation to increase the blood supply to the epiphyseal plates; growth can be slowed on the more normal side temporarily by stapling or permanently by epiphyseodesis (measures which are not free from serious complications), or the actual length of the shaft of a bone on either the affected or the healthy side can be lengthened or shortened by osteotomy. At one time it was hoped

that sympathectomy would favour acceleration of growth on the affected side, but results have not been very encouraging. The fact that a limb not only has poor circulation but also is failing to grow at the same rate as the opposite side can, however, be regarded as an indication for performance of sympathectomy in a borderline case.

No discussion of management of paralysis of the lower limbs would be complete without reference to the many devices which patients use for helping themselves to get about. *Crutches* have many designs; elbow crutches are usually preferred, but the old-fashioned axillary crutch, which fell into disrepute because of the development of "crutch palsy" can also be useful on occasions, provided the patient is warned of this risk. Quadruped and sexped *walking sticks* are light and give useful stability, while a *walking machine* running on wheels with an easy brake is useful to the patient with weak arms and trunk who cannot completely take his weight off either arm. *Wheel chair* development has improved considerably in recent years and considerable care should be taken to select the design most suitable to the patient. Particularly the various designs of lightweight portable folding chairs should be mentioned; they can be propelled by the patient's hands turning an extra rim on the wheels and have the great advantage that they will pack into the boot of most cars, or onto the back of an invalid chair. There are also useful *motor — or electrically — driven invalid chairs*; the main proviso for their use is that the patient must have reasonable power in at least two limbs, of which one must be an upper limb. The same applies also to the use of the *hand control adaptations of a motor car.*

REHABILITATION AND RESETTLEMENT

The rehabilitation and resettlement of cases with severe permanent paralysis is an integral part of the management of poliomyelitis, and one cannot regard the treatment of a case of paralytic poliomyelitis as complete until a satisfactory niche has been found for him in his family and society. The social problem of poliomyelitis has increased considerably in recent years due less to the increase of the disease than to the shift in age incidence. A disabled child is usually more readily reabsorbed than a disabled adult into his family since he is anyway in some degree dependent, and if he has no suitable home there are a variety of residential institutions and schools for disabled children. By the time he has grown up he

will have learnt to compensate for much of his disability and he will not acquire responsibilities beyond his capacity. The severely disabled adult presents a different problem. At this stage in his life he or she may have either no stable home background, or pressing family responsibilities. The need for prolonged hospital treatment and the subsequent permanent disability, perhaps with some residual physical handicap in a wage earner, or in the mother of small children, can tragically disrupt not only the patient's own life but that of his dependents and of the more distant relatives who may rally to their aid. This should be borne in mind when

for the disease to be most severely paralytic in them is overlooked

The *hospital almoner* plays an essential part in the solution of the social problems of the disabled case. She advises on financial and other difficulties caused by a patient's prolonged stay in hospital, and with her specialised knowledge of the likely outcome, she helps to plan ahead for the time when the patient comes out of hospital — still perhaps severely disabled. The patient's disability in relation to his home must be considered, and minor modifications, e g a ramp instead of steps, the placing of a handrail, the widening of a door for the passage of a wheelchair, or the provision of suitable domestic help must be arranged in good time. Alternatively it may be necessary to secure the help of the local authorities in obtaining new accommodation for the family These arrangements all need time to implement and should be considered well in advance.

Considerable tact is, of course, necessary in dealing with these

In the acute stage there is no purpose at all in trying to give a definite prognosis — unless of course disability will obviously be minimal, when unnecessary anxiety can be relieved at once. In severe cases only a guarded prognosis is justifiable; an over-optimistic one will only lead to disappointment and lack of confidence, while a gloomy one may provoke a deep-rooted disbelief which can be very difficult to deal with later. Such dramatic phrases as "he will never walk again" or "he will

again without help" are never justifiable in the acute stage; firstly, they are often wrong, and so cause needless misery; secondly, even if true, it is far better to allow realisation to come gradually, when the implications of disability are viewed in the kinder — and truer — perspective gained by seeing other patients at different stages of rehabilitation. At first the need for even one caliper may seem a tragedy; later the statement that two calipers will be required will be accepted as wonderful news because the patient is confirmed in his hopes that he may stand and perhaps even walk again. Therefore, in discussing the patient's condition when permanent disability is anticipated, one is wiser to concentrate less on considering the ultimate degree of recovery than on the fact that many muscles have been severely affected and that recovery must obviously be slow. In this way the patient and his relatives can accept that it is necessary to make arrangements for a fairly long period of hospitalisation, without taking at once the full shock of the ultimate prognosis; and modifications to the home or housing to make them suitable to the patient's return present themselves less as an acknowledgement of permanent severe disability than as a means of hastening rehabilitation. Most patients and their relatives do, in fact, by observing progress themselves, draw their own correct conclusions on the degree of possible recovery and adjust their hopes accordingly. Occasionally one meets the parent or marriage partner who will not face the obvious facts; but they are rare, and this attitude may, in a particular case, be for that individual the only possible reaction to what would otherwise be an intolerable situation.

From the resettlement aspect patients fall into three groups:

1. The *child of pre-school age*; for him it is necessary, wherever possible, to work out plans for his care at home. Ample notice should be given of his discharge so that any necessary modification in the family life can be made smoothly. It is important to induce in the family the right attitude from the start; thus the child needs extra care and attention, but at the same time he must be encouraged to gain as much independence as possible; it is also sometimes necessary to warn the parents against allowing the other children to be sacrificed to the interests of a disabled child. A child who is in hospital for long periods should be allowed to have his siblings as visitors so that they do not regard him as a stranger when finally he returns home. The same is, of course, true for any member of a

family, and children should also be allowed to visit their parents, however severely disabled, as soon as the risk of infection is past and the patient's condition is sufficiently good. It is remarkable how easily children will accept their parent's disability and chatter quite unconcernedly even to a patient in a tank respirator.

2. For *children of school age* the provision of adequate education is of prime importance since the severely disabled child will need the best education he can get to compensate for his disability. Whenever possible he should attend the school he would normally attend, and it is worth allaying the anxieties which teachers may feel regarding the dangers to a disabled child of a rough playground. Preliminary announcement to the other children that they are soon to be joined by a disabled child as normal as themselves [illegible]

Falls should be regarded as normal and be accepted without alarm by teachers and children.

For a child who is too severely disabled to be independent in feeding, dressing or toilet, or who lives too far from his local school, or whose parents cannot afford private fees, the choice lies between home education and a residential school for physically disabled children, through the Local Education Authority, the decision being influenced by both the particular home conditions and the child's abilities. As these disabled children reach adolescence their future training for employment must be considered well in advance so that time is not wasted waiting for a vacancy in the selected training centre.

3. In the resettlement of *adults* the first need is to develop the maximum personal independence; and in this the *occupational therapist* will give invaluable help Eating, drinking, shaving, dressing, page-turning, writing and typing may all have to be relearnt and the occupational therapist can contribute by helping the patient — decreasingly — and by advising and supplying the necessary gadgets required by the particular distribution of paralysis It is often astonishing how the most unpromising residual innervations can be made with perseverance to perform a few essential functions, provided the patient can have the necessary help from an experienced occupational therapist during treatment.

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The occupational therapist completes her work by helping with advice on gadgets and other apparatus for use in the home, e g. overhead runners to support the arms of a housewife in the kitchen, lazy tongs, conversion of the telephone, the correct placing of electrical switches and taps, the alteration of tap handles, and numerous other minor aids which can make the difference between partial and complete independence.

For adult patients beyond this level the question of future employment is important. Contact with the patient's former employer will sometimes provide an answer, since a different job within the patient's new limitations may be found in the same firm. This is usually the most satisfactory solution since the patient is already interested in the type of work involved, and as he recovers he may

loyment with the

onsidered. Here

Officer should be

enlisted to discover an employment which is available in the neighbourhood, is congenial to the patient and is within his physical limitations. Factors to be considered when choosing a new type of employment are not only the patient's capacity to perform the actual work, but also the likelihood of the working conditions being suitable; e.g. reasonable warmth for a sedentary patient, toilet and canteen facilities, absence of steps, and space for a wheel chair.

A preliminary period in a rehabilitation centre often helps to bridge the gap between hospital and training centre or industry; though too often these centres demand physical agility and independence beyond the capacity of patients with poliomyelitis who are none the less perfectly able to hold their own in actual employment, and there is still room for improvement in this field.

In England there are a number of training centres run by the Ministry of Labour, as well as private ventures for the benefit of the disabled. One can quote the Lord Mayor Treloar Training College at Alton, Hampshire, giving three-year courses in a variety of trades to boys aged fourteen to eighteen; the Queen Elizabeth Training College at Leatherhead, Surrey, and St. Loyes Training College in Exeter. giving three- to twelve-month courses for men and women; and the Portland Training College at Harlow Wood, Nottingham. Finally, for the patient who is insufficiently independent to be accepted at these centres there is the possibility of work at home, either independently or as a home-worker through the

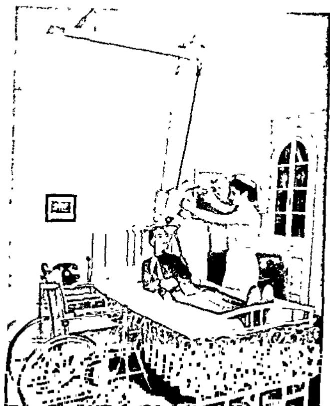


Fig 26a The Watson-Collins hoist in use. With its help a completely paralysed patient can be lifted off the bed for nursing and transferred from bed to chair or bath by one person, who attaches the webbing, winds the winch and manœuvres the patient by his feet

(Reproduced by courtesy of *Nursing Mirror*)



Fig. 26b Transferring patient to chair



Remploy Scheme. The Remploy factories themselves also take workers who cannot compete in open industry, but they are not residential. There are a few residential homes where the patient can receive some nursing care and also go out to work, but in this field there is still room for development, particularly to provide facilities [illegible] ham, Surrey; and Cripplecraft, Herne, Kent, meet the need of some patients requiring residential care as well as work in sheltered conditions.

The rehabilitation and resettlement of cases with poliomyelitis involve much ingenuity and determination on the part of the patient and his family and the staff concerned. The work is, however, rewarding, and well worth while. These patients tend always to improve rather than to deteriorate physically, pain is not a feature of their condition; they do not have to fear relapses; and they do not usually have to contend with general ill-health; they may get chilblains from the poor circulation, but this complication can be controlled (see p 125). Even ex-respiratory cases, once they have learned to manage upper respiratory infections, have remarkably little need to be off work. Difficulties in getting to and from work are rarely insuperable, and if the patient has been persistent enough to retrain and be resettled he can be expected to hold his own and to increase rather than decrease his range of activities.

Even the most severely affected patients, who are completely dependent on others can contribute quite remarkably to their families, and the children of a completely paralysed father or mother seem to thrive in the resulting closely-knit family life. Wherever possible apparatus should be provided to enable these patients to be transferred to a chair in the daytime — without physical strain on their relations (see the hoist in Fig 26) since in a wheel chair the patient's, and consequently the family's, horizon is usefully widened. In addition residual power in the upper limbs can be used optimally in the upright sitting position which is so often impossible in a bed. With steady improvement in cuirass development few, if any, patients should require to remain permanently dependent on a tank respirator, and the need for the smaller cuirass or Bragg-Paul type of respirator should not prevent a patient returning to a suitable home.

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